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## THE STATUS OF OBSTETRIC PRACTICE IN MINNESOTA\*

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THIS study was stimulated by a desire to determine how the practice of obstetrics in Minnesota fitted two conclusions of the White House Conference on Child and Maternal Welfare, called by President Hoover in 1930 and 1931.

After the Conference President Hoover promulgated the "Children's Charter" of nineteen articles, one of which was: "For every child full preparation for his birth, his mother receiving prenatal, natal and postnatal care, and the establishment of such protective measures as will make childbearing safer." There never has been such a thorough study of child and maternal welfare problems before this Conference. The fact findings and conclusions are authoritative. A few of them by the Maternal Welfare Committee will suffice to epitomize the conclusions:

1. "The United States lags behind the civilized world in the prevention of maternal mortality, three-fourths of which are due to controllable causes, for example, infection, toxemia and obstetric hemorrhages."

2. "The welfare of the mother and her offspring can only be secured by better obstetrics, and furthermore, to accomplish this, there must be better teaching of obstetrics." To us teachers this was striking close home.

3. "In 1929, in the birth registration area (all but two states) there were approximately 15,000 maternal deaths, 80,000 deaths of infants less than one month old, and 85,000 stillbirths, 65,000 of which were due to injury at birth.

4. "It is apparent that most undergraduates do not receive sufficient practical clinical training." (There were 3,600 deliveries in 1932 in the four teaching hospitals, staffed by University

teachers: Minneapolis General, University, Ancker, and Salvation Army hospitals.)

5. "There are unavoidable hazards to both mother and child, but these can be minimized by adequate teaching and practice of prenatal, natal and postnatal care, all consecutive and complete."

6. "If only our present day knowledge were universally and skillfully applied, many thousands of lives could be saved annually and much suffering and injury avoided."

*Measured by this exacting yard stick, where does Minnesota stand?*

I confess that it was with no little trepidation that this study was undertaken for I felt that if the teaching of obstetrics, at the University, has been adequate, it should be reflected in the statistics, because the graduates from the Medical School constitute about one-half of the physicians of the state.

For this study the reports of the United States Census Bureau (the last complete one being for 1929), and the reports of the Minnesota State Board of Health have been used for comparing Minnesota with the U. S. registration area and certain foreign countries.

Before making these comparisons I have thought it pertinent to discuss birth rates.

### BIRTH RATES

Birth rates, the world over, have been steadily decreasing since 1915, as is graphically shown in Figure 1, which illustrates the comparative birth curves of the U. S. registration area, Minnesota, and various European countries. In the U. S. registration area, the rate decreased from 25.1 births per 1,000 population, in 1915, to 18.9 in 1929, a drop of 25.5 per cent or more than 2 per cent a year. The Minnesota rate fell 26 per cent. Had the rate remained at its highest point, 24.7

\*Address of the retiring president of the Minnesota Academy of Medicine, January 11, 1933.

in 1915, the number of births in Minnesota, in 1929, would have been 63,000 instead of 46,713, a difference of more than 16,000 babies. In 1929, the excess of births over deaths was 21,021.

It is interesting to note also, that similar decreases are recorded in Europe: Denmark 5.6, Norway 6.1, Sweden 6.4, Italy 5.3, British Isles 7.1 and Germany 2.5 (Fig. 1).

### BIRTHS

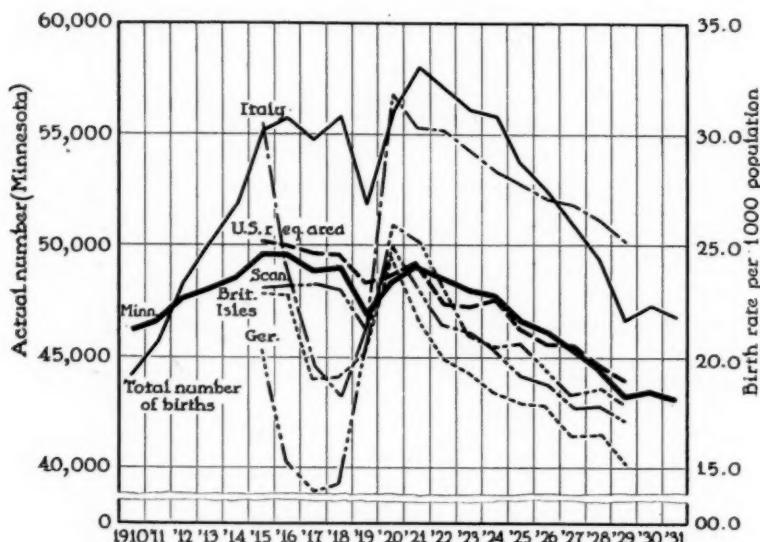


Fig. 1. Comparative birth rates of Minnesota, the U. S. registration area and certain foreign countries. The heavy black line indicates the birth rate, and the lighter black line the total number of births in Minnesota.

However, if the rate continues to decline and the total death rate remains as stable as it has for two decades, it will not be long until births equal deaths, a condition not to be contemplated with entire equanimity. It is generally estimated that when the birth rate reaches 10 per 1,000 population, or 1 per cent, then the births and deaths will be equal. This will certainly be true in this state, where the death rate has remained from 9.5 to 11 deaths per 1,000 population for twenty years.

Inasmuch as the Minnesota birth rate has remained about the same for the three years, 1929, 1930 and 1931, it may be hoped the birth rate will become stabilized before it reaches ten per 1,000 population. That similar factors are responsible for the falling birth rate throughout the country, is attested by the fact that the decrease in the birth rate in the registration area of the United States was 6.2 per 10,000 population, compared to 6.4 in Minnesota. It is inter-

### MATERNAL MORTALITY

It is often contended that because the classifications of deaths from puerperal causes differ greatly, a comparison of foreign statistics with those of the United States is impossible and unfair to this country. Methods of classifications in each country, however, change but little, therefore comparisons of increases or decreases from year to year are quite equitable. The maternal mortality rate for the U. S. registration area (all but two states) was seven per 1,000 live births, according to the Census Bureau reports for 1929 (the latest complete figures).

The Minnesota rate, 4.3 maternal deaths per 1,000 live births, is 38.8 per cent below that of the country at large. The high U. S. rate is chiefly due to the black population of the South, with a rate of twelve deaths per 1,000 live births. Excluding the negroes, the rate is 6.3. The Minnesota rate of 4.3 still shows a rate 31.7 per

cent lower than the average for the white population of the United States.

Consulting Figure 2 it will be seen that the Minnesota rate has decreased rapidly since 1920,

per cent higher than the Minnesota rate of 4.3 per 1,000 live births, although for the last decade it has a consistently lower rate.

Figure 3 graphically illustrates the story of

### MATERNAL MORTALITY (Rate per 1000 live births)

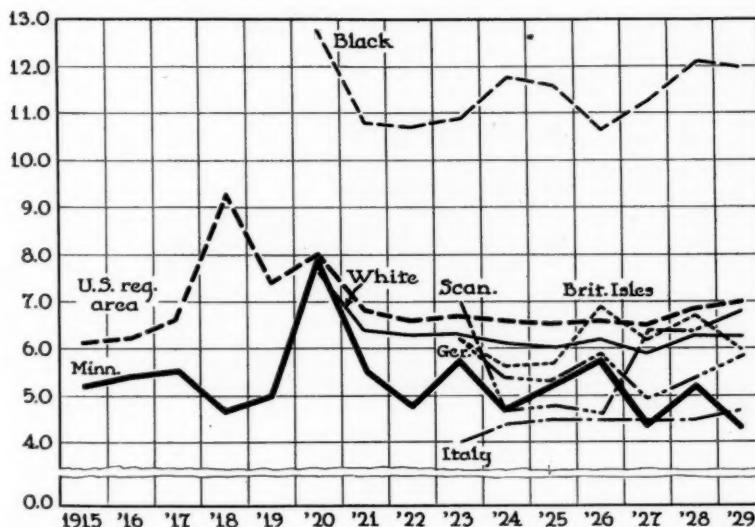


Fig. 2. Comparative maternal mortality rates. The heavy black line indicates the rate in Minnesota; the heavy broken line, the rate for the U. S. registration area. The lighter solid black line indicates the rate for the white population. The broken line, at the top, indicates the mortality rate among negroes.

while that of the registration area remains quite constant, varying only from 6.5 to 7.0 throughout the last decade, while the Minnesota rate dropped 17 per cent. Whether or not the methods of computation are comparable or fair to this country, I thought it would be interesting to compare the Minnesota rate, which is computed in the same manner as the rest of the states, with certain European countries. We have often been told that our low rate was due to our large Scandinavian population. The combined rate of Scandinavia, in 1929, was 6.3 deaths per 1,000 live births, the same as for the white population of our registration area, and the Minnesota rate 4.3, approximately 32 per cent below the "Mother Countries" as well as the U. S. registration area (Fig. 2). The Minnesota rate was also 28 per cent less than that of the British Isles and Germany, and 17.3 per cent less than the combined European average (Fig. 2). Even Italy, which had the lowest rate in Europe, 4.7, was 8

maternal mortality at the beginning and the end of the decade 1920-1929. Note the remarkable decrease in Minnesota.

#### CAUSES OF MATERNAL MORTALITY

The principal causes of puerperal deaths are the infections, the toxemias and the hemorrhages. During the decade 1920-1929, both inclusive, while the total maternal mortality rates have been progressively decreasing, the relation of these total deaths, due to each cause, has remained approximately the same, 40 per cent due to puerperal sepsis, 23 per cent to toxemias and 10 per cent to hemorrhage, and 27 to other causes.

#### PUERPERAL INFECTION

Figure 4 shows, graphically, the steady decrease of deaths from puerperal sepsis, in Minnesota (except an inexplicable rise in 1923), during the decade 1920-1929. In 1929 there were 82 deaths from infection, which gives the re-

markably low rate of 1.8 fatalities per 1,000 live births. Had the rate remained the same as it was in 1920 (2.9) the deaths from sepsis would have numbered 126 instead of 82, a reduction of

was 6 per cent: Scandinavia 33 per cent, the British Isles 14 per cent and Germany 19 per cent higher than Minnesota. Italy had a rate, for the seven years, 15 per cent better than Minnesota.

### MATERNAL MORTALITY (Last census reports)

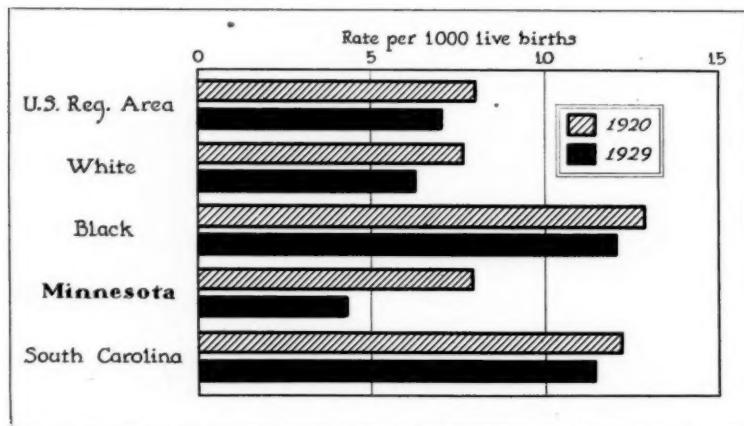


Fig. 3. Changes in the mortality rates during the decade 1920 to 1929 inclusive. The cross hatched line indicates the rate in 1920 and the solid black in 1929.

nearly 38 per cent, and a saving of 44 lives by improved aseptic technic.

In the U. S. registration area the death rate from sepsis was 44 per cent higher than in Minnesota; excluding negroes it was 33 per cent higher. The European average was 11 per cent, Scandinavia 22 per cent, the British Isles 33 per cent and Germany 61 per cent more than the Minnesota rate. In Italy the only European country with a lower rate (except Russia, which was about the same) than Minnesota, the rate was 11 per cent less (Fig. 4).

It may be argued, with some justification, that comparisons in any one year are not convincing, so I have computed the averages for the decade 1920-1929 and found that the number of deaths from sepsis in the U. S. registration area average was 19 per cent more than in Minnesota (13 per cent, excluding negroes). Complete statistics for Europe are available in the U. S. census reports only, from 1923 to 1929, inclusive. Averaging these years, the Minnesota rate is the same for the seven years as for the decade, 2.1 deaths from puerperal infection per 1,000 live births.

The average European rate for the seven years

Time and space do not permit comparisons with all European countries, so we have limited them to those which have furnished many emigrants to Minnesota. This state does not suffer, in comparison with any of the countries, usually cited as having lower rates than the United States.

Minnesota surpasses most of them, is equalled by few, and is second only to Italy and Russia.

That there has been a marked decrease, in this state, in deaths due to puerperal sepsis, may be seen graphically illustrated in Figure 4 and by the fact that deaths from this cause have decreased 38 per cent from the high point in 1920.

The average decrease in the U. S. registration area was 4.2 per cent; the European average decrease was 10 per cent. Some foreign countries have shown increases in septic deaths: Chili 143 per cent, New Zealand 125 per cent, the Netherlands 62 per cent, Australia 31 per cent, Scotland 26 per cent, England and Wales 20 per cent.

It is evident that the physicians of Minnesota are practicing cleaner obstetrics—thus approaching one of the objectives of the White House Conference.

## TOXEMIAS

Unfortunately we are unable to give comparative figures for the other two principal causes of puerperal deaths, toxemia and the hemorrhages,

comparisons. The average rate for the first decade was 1.43 deaths from toxemia per 1,000 live births and for the second decade 1.09, a decrease of 23 per cent. In 1929 the rate was 37

### DEATHS DUE TO PUERPERAL SEPSIS (Rate per 1000 live births)

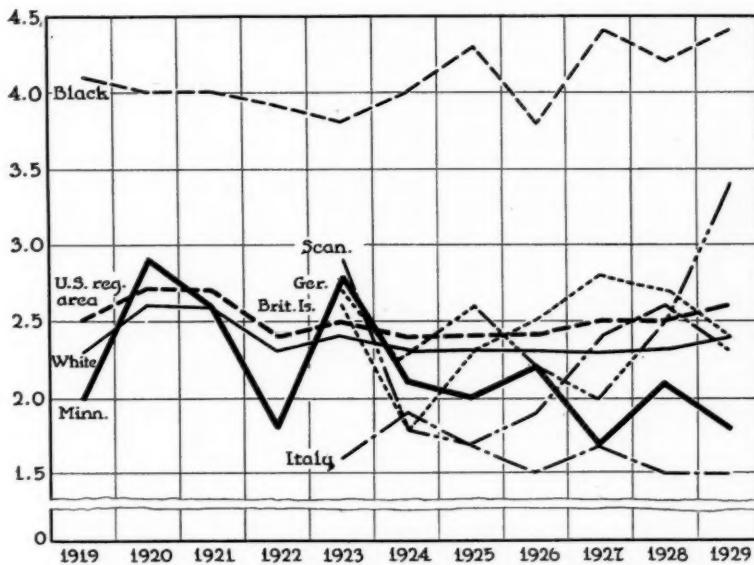


Fig. 4. Mortality from puerperal infection. The heavy black line indicates the rate in Minnesota; the heavy broken line in the U. S. registration area; the lighter solid line the rate among whites; and the short broken line the rate for negroes. The rates for foreign countries are indicated by abbreviations at beginnings of the various lines.

because the U. S. Census Bureau reports tabulate, separately, only deaths due to infection. All additional fatalities are grouped under the title "Other Causes."

Therefore we shall have to be content with an analysis of the deaths from toxemia and hemorrhage in Minnesota alone. Deaths from pre-eclampsia and eclampsia, in 1929, were approximately 50 per cent lower than in 1917, the high peak. These figures, however, hardly give a true idea of conditions, because the rates seem to fluctuate in periods of four to five years.

Figure 5 shows that there was a gradual rise from 1910 through 1913, a sudden drop in 1914, then a steady rise in 1915, 1916, and 1917, when the peak was reached, followed by a fluctuating, but generally downward tendency.

It would seem, therefore, that ten year averages would largely eliminate the indeterminable influences and give opportunity for more accurate

per cent less than for the first decade and 17 per cent lower than the second.

Evidently the physicians of Minnesota are giving increasing attention to antepartum (prenatal) care and to the conservative treatment of toxemia, the two principal methods of reducing deaths from pre-eclampsia and eclampsia,—thus approaching another objective of the White House Conference.

### HEMORRHAGE

In this state, over a period of twenty-two years, from 1910 to 1931, inclusive, puerperal deaths from bleeding have averaged 10 per cent of the total maternal mortality, less than one death per 1,000 live births.

The average number of deaths from this cause, during the first half of the period of twenty-two years, was 30.6 and during the second half 23.

The rate per 1,000 live births during the first

period was 0.58 and during the second was 0.48, a drop of 17 per cent. So we see that maternal deaths, due to all three of the principal causes, have decreased materially during the last decade

Minneapolis and 3.7 for St. Paul, in ten years, an average improvement of 43 per cent for the Twin Cities—only Oakland, Jersey City and Hartford exceeding them.

### DEATHS IN MINNESOTA FROM PRE-ECLAMPSIA & ECLAMPSIA (Rate per 1000 live births)

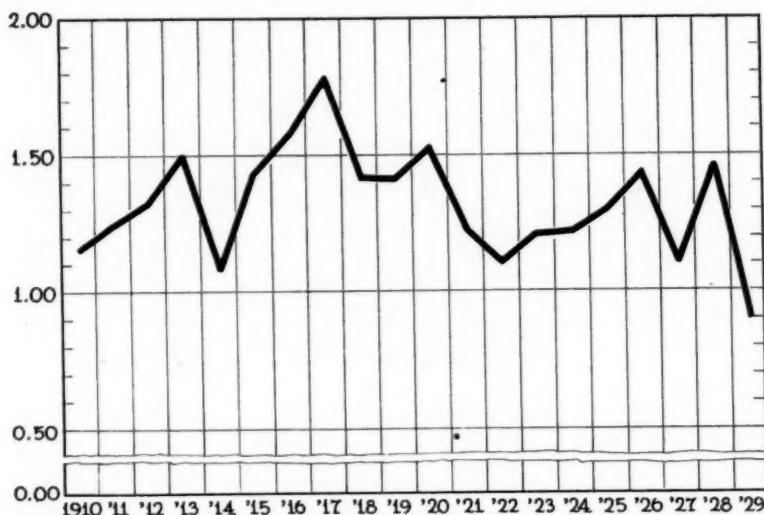


Fig. 5. Irregularly downward rate for mortality from the toxemias.

and are the chief factors in giving to Minnesota the lowest maternal mortality in the country.

It is only fair to say that several other western states, notably Iowa and Utah, have ranked very close and in some years have stood above Minnesota, but ever since the registration area has included most of the states (in 1929 all but two and at this writing all but one) Minnesota has stood at or near the top.

That this state has been improving its obstetric practice progressively is also attested by the fact that in 1920 it stood twelfth in the list, in 1928, fifth, and in 1929, first.

The Twin Cities, while a trifle higher than the whole state, also have a very high rating—Minneapolis, in 1929, standing fourth among the cities of 100,000 or over, with a rate of 4.8 (Minnesota rate 4.3). St. Paul was a close fifth with a rate of 5.0.

In 1920 the rate in Minneapolis was 8.1 and in St. Paul 8.7, an improvement of 3.3 for

### INFANT DEATHS DUE TO BIRTH INJURIES

An unhappy chapter in American obstetrics is that on deaths of infants due to birth injuries, which records that, throughout the nation, deaths from this cause have been steadily increasing at a rapid rate. Of the 85,000 deaths at birth, in the United States, 65,000 were due to birth injuries. I had hoped to find in the records that Minnesota physicians had excelled in saving babies, as well as mothers, but truth compels me to say that Minnesota is no exception to the unfortunate, death dealing tendency to hurry the termination of labor by operative means.

It will be seen by Figure 6 that the total number of infant deaths, under one year, has been steadily decreasing since 1910 (we doff our hats to our pediatric colleagues) while the actual number of deaths due to birth injuries and the rate per 1,000 live births have been rapidly rising. Had the rate, from birth injuries, remained the same as in 1910, there would have been only 80

deaths from this cause, in 1929; instead of 249, a sacrifice of 169 babies on the altar of impatience, hurry and injudicious interference with the normal processes of labor.

registration area has decreased but little, the rate in Minnesota has become the lowest in the country, dropping from 7 per 1,000 live births to 4.3, which is 38.8 per cent lower than for the country

### INFANT DEATHS DUE TO BIRTH INJURIES

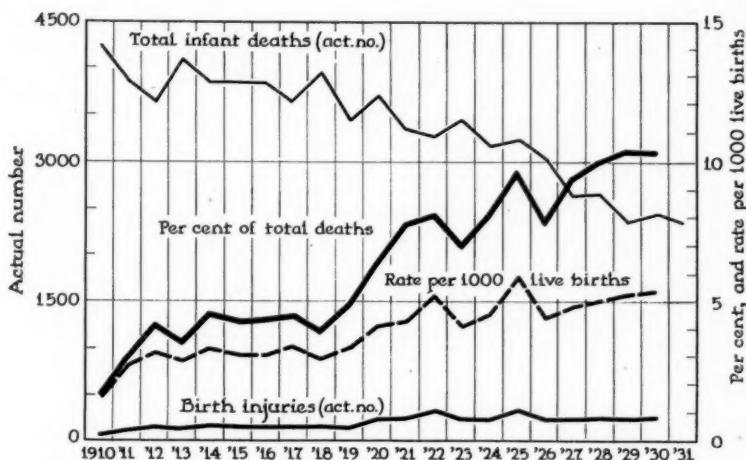


Fig. 6. Deaths from birth injuries. The heavy black line indicates the percentage of the total deaths at birth, due to birth injuries; the broken line the rate; the unbroken line, at the bottom, the total deaths, due to this cause. The light unbroken line, at the top, shows the total infant deaths, under one year.

During the two decades, from 1910-1929, both inclusive, the birth injury deaths increased 236 per cent and the rate per 1,000 live births 211 per cent.

It is difficult to understand how the profession, which has lowered the maternal mortality, so that Minnesota had the lowest rate in the nation, and lower than nearly all foreign countries, could produce such an astounding infant mortality rate from birth injuries.

In this day of decreasing birth rates, it is a social and economic necessity to save as many babies as possible, but it cannot be done if they are born unable to survive on account of birth injuries, which can only be charged to too many operative deliveries.

#### SUMMARY

The birth rate throughout the country, as well as the whole world, is falling. The Minnesota birth rate has dropped from 24.7 per 1,000 population in 1915, to 18.9 in 1929, a decrease of 26 per cent.

While the maternal mortality rate in the U. S.

at large and also lower than the European average.

The principal cause of deaths of mothers, puerperal sepsis, has, in Minnesota, shown the remarkable decrease from 2.9 in 1920, to 1.8 in 1929—33 per cent less than in the registration area, and lower than in nearly all civilized countries.

Deaths from the toxemias of pregnancy are 23 per cent below the average of the previous decade. The deaths from puerperal hemorrhages have also decreased 17 per cent.

In analyzing the results of obstetric practice in Minnesota, there seems to be only one "fly in the ointment," namely, the increasing infant deaths from birth injuries.

Taken all in all, the record of the physicians of this state, in improving obstetric practice, is quite gratifying. I am not bold enough to assert that it is due to good obstetric teaching, but I hope that may be a factor.

I feel quite confident that the work done by the State Department of Health, now under the direction of Dr. E. C. Hartley, in educating

mothers of the state about proper care, has had a real influence.

I wish to acknowledge my indebtedness to Dr. Hartley and Gerda Pearson, both of the State Department of Health, for much appreciated help in gathering the statistics necessary for this analysis.

407, University Hospital.

#### DISCUSSION

DR. R. E. SCAMMON (Minneapolis): I think Dr. Litzenberg is to be complimented on his presentation of the work being done in obstetrics. And I think the practitioners, obstetrician and pediatrician, in the state of Minnesota are still more to be congratulated.

I think it was in the early nineteen hundreds that a French obstetrician, Pierre Budin, wrote a book dealing with the care of premature and full-term infants. In 1907 that book was translated into English and the introduction was written by Sir Alexander Simpson, Emeritus Professor of Midwifery, of Edinburgh. In that introduction Simpson said: "If, in the days of the rising generation, folly should be allowed to override wisdom and the dogs of war howl down the councillors of peace, the place that France would take on emerging from a great world strife might depend largely on the strength of Budin's battalion": meaning the fighting men would be those cared for according to Budin's teachings. A war did break out as Simpson feared. In a sense Minnesota is in such a war with regard to its population and I would like to comment on Dr. Litzenberg's paper by showing you what is happening to this population of Minnesota and how important the care and preservation of the newborn is in connection with this problem.

The population of Minnesota increased rather slowly at first, then passed into a rapid phase that lasted to the nineties, and since that time has been steadily slowing down, so that now the gain in the state is only about 15,000 persons per year.

Chart 1 shows the gain in population in Minnesota, Iowa, and Wisconsin. It shows the numbers of thousands of people added to the population every year. It shows a great rise in population in the various areas with a regular but much reduced increase at the present time. A good part of these gains was due to migration, but undoubtedly some of it represents births.

Chart 2 shows the birth rate and death rate (semi-crude rates)\* in the state of Minnesota for the past ten years. It shows the decline in the birth rate that Dr. Litzenberg has described and the fairly stationary death rate. In 1920 there were 55,000+; in 1921, 58,000; in 1930 there were but 47,418 births in the state. We lose from this state about 11,000 persons each year in the exchange of population which is constantly going on between this and other political divisions. Thus the preponderance of the birth rate over the death rate becomes of increasing importance.

A part of the drop in the birth rate is undoubtedly due to the use of contraceptive measures. But it should be pointed out that the birth rate is falling in other districts very largely on account of the later marriages, where contraceptive methods are, in general, taken, for example in the south of Ireland. The birth rate also seems to tend to fall with improved social status. Another factor that we are apt to forget is constantly affecting the birth rate of this area is the change in age distribution of the population in the country. A huge number of young people pioneered this area. When

there is a stationary population, there is somewhere about 29 per cent of the population under twenty, 31 per cent or more over fifty, and 40 per cent of the people between those ages. So much of the change we see in birth rate is a perfectly natural and normal thing in an area which is not receiving young immigrants.

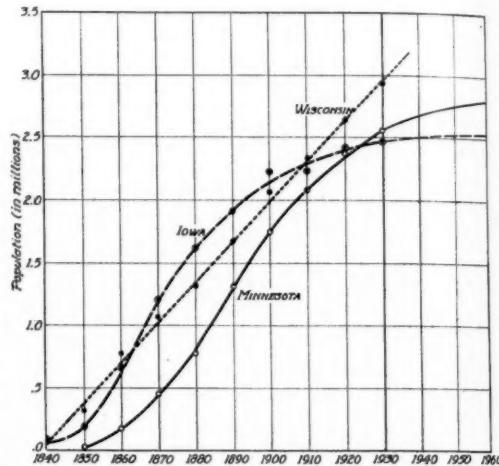


Chart 1. Three calculated curves representing the growth of population in Minnesota (A—solid line); in Iowa (B—long broken line); and in Wisconsin (C—short broken line). The curves are drawn to mathematical formulae fitted through the census observations that are indicated by various kinds of dots. The populations after 1930 are projected from these mathematical formulae and presume no extraordinary change in the character of the increase of the people of these states.

Therefore our birth rates are probably very much overestimated with respect to the future. It takes about 15 or 16 births per 1000 of population to keep the population stationary. It is probable that the population in the United States about 1965, and in Germany probably in 1945, will become stationary. I do not know when we will reach the stationary state in Minnesota; it is an extremely difficult thing to estimate; but I am inclined to think that, unless there are unprecedented social and economic changes, this static condition will be reached in a comparatively short time. I want to emphasize again with Dr. Litzenberg the importance of good obstetrical care if we are to have a population which does not decline in numbers.

The United States as a whole apparently made an increase in population of about 16 per cent in the decade from 1920 to 1930; but this increase is probably distinctly over-estimated, due to the facts that at the last census we were able to hire much better enumerators than before, that methods of tabulation have improved, and good roads and mail systems increased, so that the enumerators got in touch with people who had never been reached before. Probably the true increase was closer to 12 per cent than 16 per cent. Next to the United States comes Holland and then Belgium. The high birth rates in these little countries may be associated with the fact that both have outlets through their huge and very rich colonies. Next come Germany, Italy, Minnesota, Norway, France, the British Isles, and Sweden. The rate of increase in Minnesota is approximately that of Norway and far below that of Germany, Holland or Belgium.

I have presented these charts to show the extremely important social implications of the medical data that Dr. Litzenberg has presented this evening.

\*Semi-crude" rates are computed as the ratios of actual births and deaths to the calculated population. Since the population in Minnesota is growing very slowly, there are no significant differences between these figures and those presented by Dr. Litzenberg.

Dr. E. have been the Min cussion Dr. S

Chart year fr... its entire portion the col... calculation of each absolute loss of thousand under

Our parts: matter public This course Univer (c) w...

Dr. E. C. HARTLEY (St. Paul—by invitation): We have been trying in the Department of Child Hygiene of the Minnesota Department of Health to adjust our educational program so far as possible to the trends discussed in Dr. Litzenberg's paper and in the discussion by Dr. Scammon.

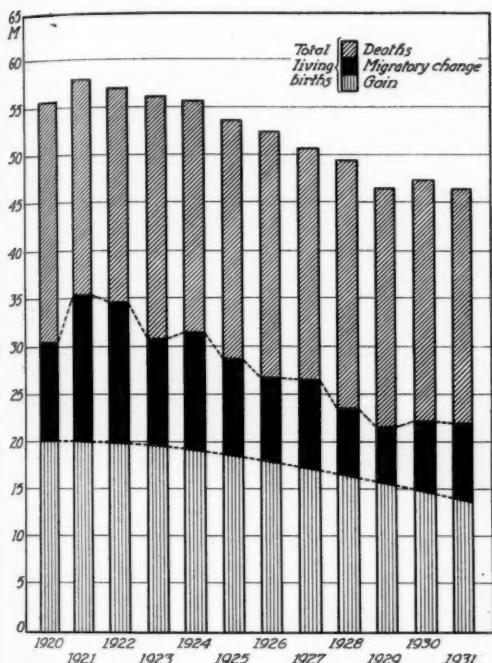


Chart 2. Changes in the population in Minnesota for each year from 1920 to 1931 inclusive. Each column represents (in its entire height) the live births of the year. The cross hatched portion of each column represents the deaths in the State during the particular year. The lower, vertically lined portions of the columns indicate the yearly gain part of the population as calculated from the population curves. And the black portion of each column indicates the excess of emigration over immigration in the State. The figure shows (a) the steady drop in absolute as well as relative number of births; (b) the invariable loss of population by migration from Minnesota; and (c) the decrease in gain of people in the State that has fallen from 20 thousand to less than 15 thousand per year in the eleven years under consideration.

Our problem may be quite definitely divided into three parts: (1) distribution of literature on the hygiene of maternity and in infancy; (2) advisory service to the public health nurses of the state; and (3) classwork. This latter is of three kinds: (a) correspondence courses given through the extension division of the University; (b) actual class work with demonstrations; (c) work for girls of eighth grade and high school age.

In this work the advantage of proper medical atten-

tion is stressed as are the standard preventive measures and accepted rules of hygiene.

The adjustments we are making to the trends mentioned consist of more carefully selecting the individuals and groups who receive this work.

An analysis of population trends shows the beginning of a falling off in the number of eighth grade girls. This reflects the rather abrupt decline in our birth rate which became apparent in 1921. Maternal deaths show relatively little change and this is in part due to the relatively larger proportion of the total births which are now primiparous births and, as such reflect the increased hazard of labor in primiparas.

With our class work at the present time we are reaching approximately 20 per cent of the girls of the state belonging to the age group of the eighth grade and about the same proportion in the group represented by the primiparous mothers. It is felt that the number will be adequate to fix communities in an intelligent and informed attitude toward the hygiene of maternity and infancy and toward the contributions which the medical profession has to offer in this portion of its field.

DR. HOBART JOHNSON (by invitation): I am sure I have nothing to add to what has been said this evening except that I believe our black man has one point on the white community. Dr. Litzenberg said it was the pediatricians who had been urging the obstetricians to do this antenatal work. In Africa where I have been, supposing the mother dies in childbirth, the husband must pay a heavy fine to the brothers and sisters of the deceased. So far as I know our tribe is the only one which has this custom. I thought it was something we might build on when we begin stressing on antenatal care; just put it up to the husband. Among our black people over 50 per cent of the babies die within the first year; how many die within the first month I do not know. There is no registration there.

Another thing I might mention. So far as I know there is no eclampsia among our people. We have never seen a case and our usual run of cases at the dispensary is from 100 to 300 a day—not, however, in obstetrics alone. There are also surprisingly few cases of puerperal sepsis; why it should not be almost 100 per cent, I don't know, but puerperal sepsis is exceedingly rare though there is not the slightest sense of aseptic technic—indeed there is everything but that.

DR. LITZENBERG (closing): I have nothing particular to add except to thank Drs. Scammon, Hartley and Johnson. Dr. Scammon has hit the nail on the head by drawing attention to certain fundamental trends which bear upon birth rate. We also have a condition which I have not discussed tonight and that is contraception. Our birth rate is more or less rapidly approaching the death rate. That puts it squarely up to the departments of obstetrics and pediatrics to train these young men so that the number of conceptions when they do occur shall result in living babies. The work which the pediatricians have done in this country has been marvelous in saving the babies. We can't do much to alter these fundamental trends. We can't do much about contraceptives; when people do not want babies they are not going to have them. But we can do something to save those who will conceive.

## LEUKEMIA AND ITS TREATMENT\*

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*Bismarck, North Dakota*

LEUKEMIA was recognized by Hughes Bennett<sup>1</sup> in 1845 and his clinical description was followed by the report of Virchow a month later. Bennett described it under the name of leukocytopenia as "a suppuration of the blood attended by enlargement of the spleen and liver." Virchow described it under the name of leukemia or white blood. In 1870 Neuman<sup>11</sup> determined the importance of changes in the bone marrow. The work of Ehrlich,<sup>4</sup> in 1885, enabled one to classify the cases according to the blood changes.

This disease is characterized by the appearance of immature leukocytes and usually by an increase in the normal leukocytes in the blood associated with a primary hyperplasia of the leukopoietic tissues, either myeloid or lymphadenoid, throughout the body. Two distinct types are recognized clinically; (1) the myeloid type characterized by the hyperplasia of the white cells of the marrow (granulocytes) and the appearance of myelocytes to the extent of 20 to 40 per cent of the total leukocytes present in the blood; (2) the lymphatic type in which 80 per cent and over are lymphocytes.

### ETIOLOGY

No special cause has been found although recent workers have strengthened the theory that infection plays an important rôle in the production of the disease, and claim that certain micro-organisms have been found. Some experimenters have succeeded in conveying chicken leukemia from one chicken to another; similar experiments upon men and ordinary laboratory animals have been unsuccessful. Most of the medical men have been inclined to believe that leukemia is a malignant disease, because of its course and characteristics similar to other malignant diseases.

### CLASSIFICATION AND COURSE

Acute lymphatic leukemia occurs most frequently during the first five years of life and in the second decade. After the age of twenty-five its incidence is greatly diminished, and occurrence after sixty years of age is rare. The aver-

age duration is from a few days to three weeks.

Chronic lymphatic leukemia usually occurs between the ages of forty and sixty. The average duration varies from four months to five or six years.

Acute myelogenous leukemia is fairly rare. In 1904 Hirschfield<sup>6</sup> found only six undoubted cases. In 1917 Gorham<sup>5</sup> reviewed the literature and could find only twenty-eight autopsied cases of acute myelogenous leukemia. The average duration of the disease was from a few weeks to three months.

The type of chronic myeloid leukemia occurs usually between the ages of twenty-five and forty-five.

The sex predominance in leukemia is about two males to one female.

### CLINICAL SYMPTOMS

The most prominent symptoms of leukemia are enlargement of the spleen and adenopathy involving any or all chains of lymphatics. The other symptoms may be melena, loss of strength, undue fatigue, loss of weight, pain, gastro-intestinal disturbance, hematemesis, hemoptysis, epistaxis, dizziness, syncope, and headaches.

### PATHOLOGY

Briefly, in lymphatic leukemia the glands on section appear soft, gray or pinkish in color, smooth, and homogeneous. The tissue is packed with lymphocytes, mostly the small type, not only filling the vascular space, but infiltrating the surrounding tissue.

The spleen shows decided enlargement; the surface of the spleen is smooth and the normal outline preserved. The substance of the organ is filled with infiltrating lymphoid cells and the microscopic structure is almost unrecognizable. The bone marrow is dense and homogeneous, and is gray or grayish red in color. Microscopically, there is almost entire replacement of erythoblastic and myeloblastic tissue by lymphoid cells. The liver is enlarged, and the development of lymphocytic perivascular nodules in the connective tissue is characteristic.

\*From the Quain and Ramstad Clinic, Bismarck, North Dakota.

In chronic myeloid leukemia the bone marrow is pink or yellowish gray, and is firm in texture. The tissues are crowded with leukocytes and erythocytic cells, but the chief feature of this intense hyperplasia is the myeloid cell. The spleen is greatly enlarged; it may weigh from two to twenty pounds, and the normal outline is preserved. It is firm and has a grayish tint and cuts with difficulty, due to the great increase of fibrous tissue in the stroma. There is a large number of cells in the tissue, chiefly the myelocytic forms. Hemorrhage and infarct are not uncommon. The liver is enlarged and the capsule contains myeloid cells.

#### TREATMENT

In the acute types of leukemia the treatment is purely symptomatic. Isaacs<sup>9</sup> reports that eighty-four out of 113 patients suffering from acute leukemia died within two months.

Arsenic had been the favorite drug used in the treatment of this disease before benzol and radiation came to be used. Its administration in some cases caused apparent or actual arrest of symptoms, or remissions. One should try, however, some other agencies first, because frequently arsenical poisoning has occurred.

Splenectomy has been employed in leukemia, but is unjustifiable because the patients all die from the operation, either from primary or secondary hemorrhage.

Benzol as treatment for leukemia was first introduced in 1912 by Koranzi,<sup>8</sup> who observed three female patients who had been poisoned by benzol from the material upon which they were working. These women developed severe aplastic anemia; the white and red cells were tremendously reduced. Upon these observations, Koranzi reports one case of chronic myelogenous leukemia where the white cells dropped from 173,000 to 12,000 after three and one-half months treatment. The patient was in fairly good condition.

In 1912 Kiralyfi,<sup>7</sup> a Hungarian, treated seven cases of chronic myelogenous leukemia with benzol, the length of treatment varying from three to five months. He reports that he obtained normal counts and the patients were symptom-free. Kiralyfi, however, did not report how long his patients were symptom-free. No one seems to be able to duplicate his unusual results.

In 1912 Steen reported a case in which the pa-

tient was treated by x-ray and was well for two months. He was then treated with benzol and the white cells fell from 225,000 to 6,000 after forty-two days' treatment.

In 1917 Winslow and Edwards<sup>14</sup> tried benzol in one case. The white cells dropped from 550,000 to 46,000. They observed that there were some symptomatic results, marked gastric irritation, great danger of benzol poisoning, and too rapid decrease of white cells.

Billings,<sup>2</sup> in 1913, reported four cases of leukemia of the myeloid type. One patient had previously been treated with x-ray, excellent results having been obtained, but on account of a severe x-ray burn, this treatment had been discontinued. Benzol was then administered with good results. His other three patients were treated with benzol and x-ray with equally good results. He also reported one case of chronic lymphatic leukemia where x-ray and benzol caused the white cells to fall from 450,000 to 4,900, and the patient showed marked improvement in his condition. Although Billings was enthusiastic about benzol treatment, his report showed that he considered x-ray as an essential part in the treatment.

H. Pancoast,<sup>12</sup> in 1917, reported four cases of leukemia in which benzol was tried. One patient whom x-ray no longer benefited responded to benzol and lived an additional three months. Two of the patients did not respond to benzol, but did to x-ray. The fourth patient was receiving benefit from x-ray, but remained away for a year and a half without being checked up and had a relapse. He was improved under x-ray, but during the hot weather had another relapse and x-ray had to be discontinued. Benzol was tried without any success.

Boardman,<sup>3</sup> in 1916, reported the reduction of the white cells from 202,000 to 18,000 and the lymphocytes from 98 to 77 per cent in one case of chronic lymphatic leukemia with the combined use of x-ray and benzol. Another patient with chronic lymphatic leukemia under benzol treatment alone for one month did not show any improvement. His third patient, suffering from chronic myelogenous leukemia, was under treatment with x-ray and benzol for three months. The last report, five months later, showed that the man was in comparatively good health.

X-ray was first considered as a treatment for leukemia in 1902. Because of the marked development of x-ray technic in the past ten years,

x-ray is bound to take the leading position in the treatment of leukemia. X-ray treatment alone has been proven to be very valuable in leukemia. Minot<sup>10</sup> collected 148 cases from 1898 to 1923 of chronic myelogenous leukemia. The average duration of the disease from the onset of the symptoms, before a definite diagnosis was made, was 1.4 years. He then reported seventy-eight cases of chronic myelogenous leukemia which had received irradiation and the patients lived on an average of 3.5 years after the diagnosis was made; in fifty-two cases not treated by irradiation, the average duration after diagnosis was 3.04 years. Fifty per cent of the seventy-eight treated patients improved sufficiently from a condition of distinct ill health, sometimes bedridden, to a state of comparative well-being. Less than 5 per cent failed to improve to a degree of health that rendered the patient able to do some light work. Definite remissions of moderate degree have occurred in four (7.7 per cent) of the fifty-two patients now under radiation.

In 1924 Minot and Isaacs<sup>9</sup> reported eighty patients with chronic lymphatic leukemia over thirty years of age. Thirty received no irradiation and fifty did. Irradiation did not prolong life. The average duration of the disease in both untreated and treated cases from the time of diagnosis was 3.5 years. Minot also found that the average duration of the disease from the first symptoms until definite diagnosis was made was 1.4 years. He was able to tabulate sixty-one cases in which x-ray had been used. No improvement occurred in twenty-three per cent, rather trivial improvement in 30 per cent, moderate or distinct improvement in 47 per cent, and 10 per cent showed marked improvement. The chances for improvement decreases as the disease progresses, and as time passes x-ray becomes less and less effective. Spontaneous remissions of slight degree occurred in only 5 per cent of the 36 non-irradiated patients, while 57 per cent of the sixty-one patients radiated showed distinct improvement. The patient's improvement is manifested by his sense of well-being. The reasons for these results are that x-ray decreases the most prominent symptom of abnormal fatigue; it lessens the anemia by increasing bone marrow function; it decreases the activity of the lymphatics and lessens the mild or marked symptoms due to pressure from the enlarged internal or external glands.

Deep x-ray therapy was used exclusively in our Clinic. We did not attempt to treat the acute cases, since the prevailing opinion is that these patients do not respond to x-ray treatment. In the chronic cases we radiated the enlarged glands, enlarged spleen and the long bones. The initial dosage used was about one-fourth to one-half of an erythema dose to one or two areas. Further treatment and dosage were guided entirely by the white count. With a rapidly falling count the treatment was stopped when the white count reached 40,000 to 50,000. If the count was decreasing slowly, the treatment was stopped when the white count reached 20,000 to 30,000. An attempt was made to reduce the glands to normal size in two or three treatments, but this was guided entirely by the white count. The patients reported monthly for a white count and radiation, if needed. They usually reported that they felt better and many were able to return to their former occupations.

#### SUMMARY OF TREATMENT

The treatment of acute leukemia is purely symptomatic.

Extensive use of arsenic in leukemia has been discontinued because of the great danger of poisoning. Splenectomy only hastens the death of the patient. Benzol has been found to be of value in the treatment of leukemia by Koranzi and Kiralyfi only, but they failed to publish the duration of the benefits derived from this form of treatment. Their work has not been duplicated with the success that they claim. Other benzol enthusiasts, like Boardman and Billings, have had to use the x-ray in conjunction with benzol. Minot, who has done more work on x-ray therapy in leukemia, has definitely proven that x-ray is the most valuable form of treatment. Benzol is accompanied by great danger of poisoning, too rapid a decrease of the white cells and in a majority of cases a marked destruction also of red blood cells. It produces, at least, considerable discomfort from gastric disturbance. It cannot be administered intravenously because it causes death, nor subcutaneously because it causes necrosis, and given by rectum it causes marked irritation and cannot be retained. It also is a very difficult drug to control. X-ray under competent men has been proven to be easily controlled, is not dangerous, and it lessens anemia.

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have been authentically duplicated in all parts of the country, the x-ray cannot be replaced by any other method now in use, and assumes the leading place in the treatment of chronic leukemia.

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## AGRANULOCYTOSIS\*

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DURING the past ten years over three hundred cases have been reported of a severe, frequently fatal illness with a peculiar blood picture, *viz.*, a profound leukopenia with total or almost complete absence of the granular polymorphonuclear cells. Described by Schultz<sup>4</sup> in 1922, this disease, agranulocytosis, presents several distinctive features: a striking predilection for women of middle age or over; sudden onset; marked prostration; high fever; necrotic ulcerative lesions of mucous surfaces, especially the mouth, throat and anus; disappearance of the myeloid or granulocytic elements from the bone marrow, with a corresponding leukopenia and granulopenia in the blood stream; and a rapid, frequently fatal course.

Particularly constant in the agranulocytosis of Schultz are three major characteristics: (1) the practically complete disappearance of the cells of the myeloid series from the bone marrow without demonstrable damage to the erythropoietic system or to the megakaryocytes; (2) the markedly low white cell count with an extreme granulopenia and lymphopenia without hemorrhagic diathesis; and (3) the ulcero-gangrenous lesions in the mouth and genito-anal areas. These three features may be said to correspond to three distinct "onsets," or stages, of the disease as postulated by Roberts and Kracke,<sup>5</sup> *viz.*, a bone mar-

row onset; a blood stream onset; and a clinical onset. The first stage is confined to the bone marrow and may be looked upon as the prodromal period, being represented clinically by fever and prostration; the second stage is manifested in the blood stream by a marked leukopenia and granulopenia; while the third stage is represented by ulcero-gangrenous lesions, collapse, and frequently death.

The etiology of agranulocytosis is unknown, but the primary site of insult in the disease is undoubtedly in the bone marrow which microscopically shows a complete or nearly complete disappearance of the cells of the myeloid series. Rapid regeneration of these cells, however, with a corresponding increase of the granular leukocytes in the blood stream can take place with recovery or with a period of temporary improvement. This can be clearly demonstrated at autopsy or by biopsy, as well as by an increase in neutrophils in the leukocyte counts during recovery or improvement.

The necrotic ulcerative lesions of agranulocytosis occur most commonly on the tonsils, which at first are enlarged and reddened but soon become covered with a greyish or yellowish exudate which may cause sloughing of the involved areas. Other parts of the oral cavity—the pharynx, uvula, soft palate, gums and tongue—may be the site of involvement. Identical lesions

\*Read before the Minnesota State Medical Association, Saint Paul, May 24, 1932.

may also develop in and about the external genitalia and anus. The gastro-intestinal tract, particularly the esophagus, may be extensively involved. Bullous cutaneous lesions may, in addition, be occasionally present. The typical mucous membrane lesion of the disease is characterized first by an intense local exudative inflammation not unlike that caused by diphtheria. This is soon followed by an ulcer-gangrenous process which often causes sloughing of the involved mucous membrane, producing additional or multiple ulcerative lesions of identical appearance. These may coalesce to produce an irregular, extensive lesion, or there may be isolated ulcers on various parts of the mucous surface. The base of the ulcer is usually filled with a thick, yellowish or greyish necrotic exudate which emits a strong, fetid odor. Microscopically the lesion shows deeply extending necrosis without definite separation from the surrounding normal tissue and exhibiting no marked cellular reaction or neutrophilic infiltration.

The following cases illustrate typically the various manifestations of agranulocytosis.

**Case 1.**—A white woman, aged fifty-three, was brought to the Miller Hospital on November 26, 1931, in extremis. Her past history revealed the following important features: an unusual susceptibility to severe "colds"; a leukocyte count of 6,400 with 50 per cent polymorphonuclears in 1924; an attack of acute thyroiditis in January, 1931, with a basal metabolic rate of minus 30 per cent, which improved under thyroid extract; and a leukocyte count of 4,300 with 40 per cent polymorphonuclears in April, 1931. The history given of her present illness was that on November 16, 1931, she had developed a sore throat with pain on swallowing. A patchy grey membrane had then appeared on the tonsils, cultures from which proved negative for diphtheria. Her temperature had averaged between 101° and 102°. Weakness and prostration were profound, and the course of the disease had been rapidly downward.

On admission to the hospital she was in a deep stupor. The mucous membranes of the mouth and pharynx were red and swollen. The tonsils were covered with a dirty greenish necrotic exudate. The tongue was greatly swollen and showed numerous pin-head areas of necrosis. Two leukocyte counts showed the total number of cells to be only 200 and 150, respectively, with complete absence of polymorphonuclear cells. The hemoglobin was 67 per cent; red cells 3,760,000. Blood smears showed a normal number of platelets. Death occurred a few hours after admission, ten days from the onset of the first symptoms. No autopsy was obtained.

**Case 2.**—A white woman, aged fifty-eight, who had complained for two months of tiring very easily and

of swelling of the ankles with shortness of breath, suddenly developed a sore throat with marked weakness and fever on March 1, 1931. A year before, an examination which she had undergone had revealed a systolic blood pressure of 230, an enlarged heart, and a leukocyte count of 6,000.

When first seen on March 4, 1931, three days after the onset of her acute symptoms, the throat was dif-

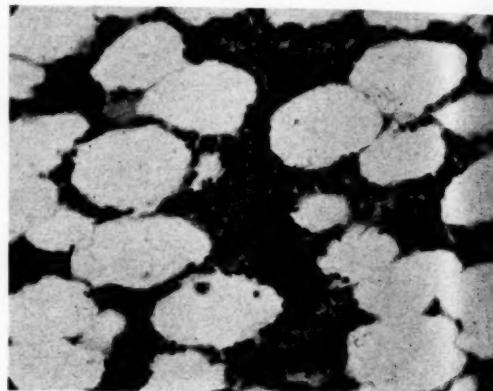


Fig. 1. Bone marrow from Case 3, showing absence of granulocytic elements.

fusely inflamed, prostration was extreme, temperature 100.8°, pulse 110, leukocyte count 1,200, hemoglobin 40 per cent, erythrocytes 2,000,000. The urine contained albumin and casts. On March 5 the leukocytes were 800 with a complete absence of granular cells, lymphocytes comprising 95 per cent, monocytes 5 per cent. On March 6 the leukocyte count had still further dropped to 340, death occurring a few hours later, five days after the acute manifestations of her illness began. Autopsy showed hypertrophy of the heart (weight 390 gm.); hypostatic pneumonia; and an "apparently normal" bone marrow.

**Case 3.**—This case, published through the courtesy of Dr. J. M. Hayes and Dr. M. Barron, is that of a white woman, aged sixty, who was admitted to St. Mary's Hospital on November 17, 1931. She had always been previously healthy but had recently been undergoing treatment for a sinusitis when, on November 11, 1931, she developed a sore mouth with headache, nausea, vomiting and fever. On admission to the hospital, prostration was marked, temperature 104°, pulse 100. Small discrete ulcers were present on the mucous membrane of the lips, mouth, palate and pharynx, with an impetiginous eruption over the lower lip. The total leukocyte count was 500, with a complete absence of polymorphonuclear cells. The hemoglobin was 67 per cent, erythrocytes 3,700,000. The urine showed one plus albumin. Examination was otherwise negative.

During the first twenty-four hours in hospital she was given two transfusions. Following these the leukocyte count rose to 1,000. Two days later the leukocyte count was 600 in the morning and 300 in the after-

noon with no polymorphonuclear cells present. Two more transfusions were then given without any improvement. The leukocytes dropped to only 100 cells. The temperature rose to 105°, pulse to 135, and respirations to 50. Death occurred on November 21, ten days after the appearance of the first symptoms.

Autopsy showed a red bone marrow containing no granulocytic elements (Fig. 1); a large soft spleen;

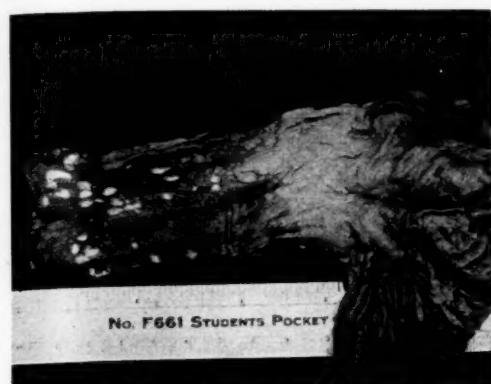


Fig. 2. Extensive ulceration of esophagus in Case 6.

broncho-pneumonia; an enlarged heart (400 gm.); and a soft friable liver.

**Case 4.**—A white woman, aged thirty-four, who had been acutely ill for a week with a high fever, extreme weakness, and pain in the rectum, had a thrombotic hemorrhoid excised in a physician's office under local anesthesia on May 16, 1930. Immediately following this she collapsed and was removed to St. Luke's Hospital, where she remained twenty-four hours. The leukocyte count on this admission was 1,000, the hemoglobin 47 per cent. Two days later she was readmitted on the service of Dr. Warner Ogden, through whose courtesy the case is published. On examination the site of the hemorrhoidectomy was covered with a dirty, greyish, membranous exudate which emitted a foul odor and showed Vincent's organisms in abundance in smears. Prostration was extreme. In the blood examinations the outstanding feature was a marked leukopenia with complete absence of polymorphonuclear leukocytes, all of the cells being lymphocytes of the mature type. Leukocyte counts made daily ranged from 550 to 1,000 with no granular cells found on any examination. Hemoglobin estimations averaged 50 per cent, red cell counts 4,500,000. The course of the disease was rapid, marked by extreme prostration, chills, temperatures of 103° and 104°, and a high pulse rate. Death occurred on May 24, fifteen days after the onset of the first symptoms.

**Case 5.**—A white woman, aged sixty-five, developed an "acute cold" with a sore throat on January 26, 1932. In the course of the next few days she became very weak and her throat grew steadily more painful. When first seen, one week after the onset of the initial symp-

toms, she was in a state of extreme collapse with a pulse rate of 140 and a temperature of 104°. A marked exudative inflammation with a strong fetid odor involved the entire throat, and moist râles were present throughout both lungs. Immediate administration of antistreptococcal serum failed entirely to result in any response, death occurring a few hours later. At autopsy the chief findings were: large numbers of megaloblasts with absence of granulocytes in bone marrow smears; enlargement of the heart (wt. 450 gms.); diffuse congestion of the lungs; and a growth of hemolytic streptococci in cultures from the spleen and throat.

**Case 6.**—This case, published through the courtesy of Dr. Alfred Hoff, is that of a white woman, aged sixty-four, who was admitted to St. Luke's Hospital on March 23, 1929, with a fluctuating painful swelling which had been present for twelve days below the angle of the left jaw. The tonsils were enlarged and reddened, the temperature 101°, and the leukocyte count 3,500. Following the establishment of drainage the suppurative adenitis healed uneventfully with a prompt return of the temperature to normal.

One month later an upper molar tooth was extracted and within twenty-four hours the gums, the throat and the palate had all become extremely swollen and painful. Two days later she was readmitted to the hospital in a state of profound prostration. The gum at the site of the extraction was now gangrenous. Adjoining it was a deep ulcer covered with a thick, grey, tenacious exudate, while all of the surrounding gum tissue was red, swollen and spongy. Smears showed Vincent's organisms, and a non-hemolytic streptococcus grew in cultures from the exudate. The leukocyte count was 4,300 with only 4 per cent polymorphonuclears. The hemoglobin was 70 per cent, red cells 3,500,000.

Progress was exceedingly slow. Weakness was extreme. The lesions on the gums gradually healed but another painful stubborn ulcer developed in the rectum. There was a daily fever ranging from 99.5° to 102°. A constant leukopenia was a striking feature, the leukocyte count averaging 3,800 (20 counts), the lowest being 1,360. The polymorphonuclears were continuously low, their average being 8 per cent, the remainder of the white cell differential being made up of 79 per cent lymphocytes, 13 per cent monocytes. No anemia developed (hemoglobin 75 per cent, red cells 4,000,000). Platelets 120,000. Bleeding and coagulation times normal. Blood cultures negative.

After five weeks in the hospital she was discharged improved on June 8, 1929, and during the next two months continued to improve slowly but steadily, gradually resuming her full activities. Complete healing of the rectal ulcer finally occurred, and the blood count returned to an absolute normal.

On August 6, 1929, she developed a sudden pain in the right chest, with vomiting and tarry stools. Signs of consolidation appeared rapidly in the lower lobe of the right lung, and in forty-eight hours she was comatose. Death occurred on August 9, 1929. A blood examination on the day of her death gave the following figures: hemoglobin 90 per cent, erythrocytes 4,500,000;

leukocytes 7,000 with 76 per cent polymorphonuclears, 22.5 per cent lymphocytes, 1.5 per cent monocytes. Platelets were plentiful in the smears.

The findings at autopsy were as follows: Esophagus: walls thickened and edematous; mucosa diffusely ulcerated, the base of the ulcer being of a dark purplish color with only a few small scattered islands of swollen mucosa remaining (Fig. 2); microscopically there

out a blood examination the diagnosis of agranulocytosis can be made on the bone marrow findings with the clinical triad in a woman, of fever, prostration and mucous membrane lesions. In Case 6 it is most probable that the ulcerative lesions in the esophagus and bronchi were originally present during the acute attack of agran-

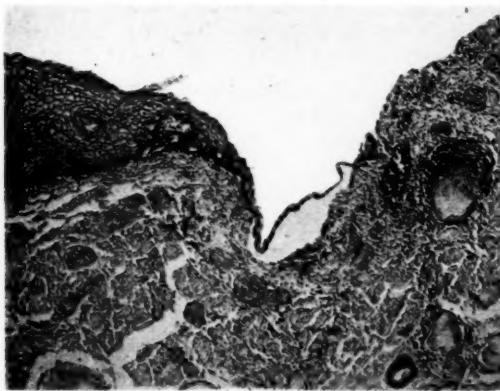


Fig. 3. Microscopic appearance of the esophageal ulceration in Case 6.

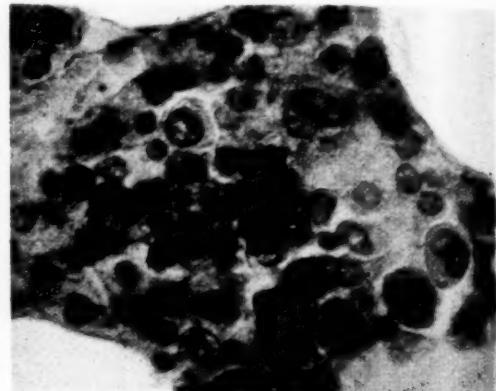


Fig. 4. Bone marrow in Case 6, showing active hyperplasia of all elements.

is extensive ulceration of the esophageal mucosa with areas of epithelial thickening and regeneration, the base of the ulcer showing dense fibrosis and marked congestion with patchy infiltration of a few leukocytes (Fig. 3). Lungs: the main bronchi show patchy exfoliations of the epithelium and areas of extensive ulceration with fibrosis and congestion, with infiltrates in varying numbers along the base of the ulcers; the lower lobe of the right lung shows thrombosis of the main artery with hemorrhagic infarct of the involved areas and patchy infiltration of polymorphonuclears throughout. Heart: 425 grams, left ventricular hypertrophy. Terminal ileum and colon: diffuse edema and congestion of mucosa. Kidneys: cloudy and swollen; right, 165 grams; left, 145 grams. Bone marrow of right femur: red meaty tissue in excess amount showing microscopically active hyperplasia of all elements (Fig. 4).

#### DISCUSSION

The close similarity in all of the findings in these six cases of agranulocytosis is very striking. The sudden onset in women of an acute, rapidly progressive illness with fever, prostration and necrotic ulceration of mucous surfaces, together with a profound leukopenia and a complete or almost complete absence of the polymorphonuclear leukocytes, form a syndrome which is most distinctive. In Case 5 even with-

ulocytosis three months before death, and that they persisted during the period of prolonged convalescence. This patient actually had never fully recovered from the attack and the lesions must have remained more or less active during the period of supposed recovery. It is reasonable to believe that the pulmonary complication which caused death in this case was brought about by the acute activation of the ulcers, particularly of the bronchi. The presence of neutrophils in the exudate, indicating return of leukopoiesis in the bone marrow during convalescence, as is evidenced, too, by the return to a normal leukocyte count, is of particular interest in Case 6.

Failure of the myeloid element in the bone marrow is the fundamental feature in agranulocytosis. With the resultant withdrawal of the leukocytes from the blood stream, the chief barrier of defense against bacterial invasion is down, and unless a rapid reappearance of granulocytes occurs, a fatal outcome, preceded by collapse and sepsis, is inevitable. The underlying basis for therapy in agranulocytosis, therefore, logically consists in the use of measures directed toward the stimulation of the inactive bone marrow. Of

the numerous agents employed for this purpose, the recent addition of pentose nucleotides,<sup>2</sup> marks the greatest advance in the treatment of the condition.

Agranulocytosis, instead of pursuing an acute course, may in rare instances occur in a chronic recurrent form. The following case is an example of this type:

*Case 7.*—A white woman, aged forty-eight, was admitted to the Miller Hospital on January 12, 1932, because of weakness, fever, swelling and ulceration of the lips, tongue and gums, ulcers of the rectum, and loss of weight. The present illness had started two years before with a severe sore throat, fever and weakness, which had kept her in bed for a month. Since then, she had had four similar attacks. Bone marrow administration and x-ray treatments to the long bones had apparently had no influence on the course of the disease. Her present sore mouth and throat had begun a week before admission, and, in addition, she had developed painful anal ulcers with severe diarrhea. She was so prostrated that she could hardly move because of the extreme weakness. Her family history was negative.

On examination, the lips and tongue were red and swollen, and presented several round, punched-out ulcers, the size of a pea and of a grayish color, also numerous small white scars from previous ulcers. The tonsils and pharynx were clear. The heart and lungs were normal. Blood pressure, 100 systolic, 66 diastolic. The spleen was enlarged and firm, the lower pole descending 5 cm. below the costal margin. The rectal and anal mucosa was swollen and red, and proctoscopic examination revealed several chronic ulcers with much inflammatory reaction, which bled easily and were very painful. The leukocyte count on admission was 800. The following day the leukocytes were 1,050 with 12 per cent neutrophils and 85 per cent lymphocytes, hemoglobin 51 per cent, erythrocytes 3,310,000. The temperature ranged from 99° to 102.2°.

The progress of the case during her hospital stay of thirty-four days was not encouraging. The mouth and rectal lesions slowly healed but the size of the spleen increased and free fluid developed in the abdominal cavity. The blood picture remained essentially unchanged, a marked leukopenia and granulopenia being constant. Bone marrow feeding had no influence on the blood counts. Various investigations, including roentgen evidence of any gastro-intestinal lesion, search of the stools for amebæ, and blood cultures, were all negative. In spite of the evidence to the contrary on the part of the abdominal findings and the blood picture, with the healing of the mucosal ulcers, the patient insisted she was very much improved and left the hospital on February 16, 1932, with a leukocyte count of 550.

The leukocyte counts over a two-year period in this remarkable case are shown in Table I.

TABLE I. AGRANULOCYTOSIS (CHRONIC, RECURRENT)

	Case 7	
Date	Total W.B.C.	Granulocytes
Jan. 31, 1930	2,040	14
Feb. 3	2,100	28
Feb. 4	1,740	
Feb. 5	1,950	
Feb. 8	2,000	
Feb. 13	2,300	
Feb. 15	1,840	
Feb. 21	1,600	36
Mar. 6	1,100	53
Aug. 5	1,250	
Oct. 2	1,000	14
Oct. 29	1,100	17
Nov. 3	1,275	20
Dec. 16	2,150	29
Aug. 25, 1931	1,840	22
Nov. 17	711	
Nov. 29	644	20
Nov. 30	1,460	
Dec. 1	3,760	
Dec. 2	1,020	
Dec. 3	1,140	
Dec. 4	1,100	
Dec. 5	754	
Jan. 12, 1932	1,050	12
Jan. 13	750	16
Jan. 16	500	
Jan. 18	500	34
Jan. 19	850	
Jan. 20	525	
Jan. 21	900	
Jan. 22	750	
Jan. 23	1,175	
Jan. 25	1,650	59
Jan. 26	1,350	
Jan. 27	850	
Jan. 28	1,250	
Jan. 29	1,000	
Jan. 30	1,000	
Feb. 2	850	
Feb. 3	925	
Feb. 4	950	
Feb. 5	875	46
Feb. 6	825	
Feb. 8	850	
Feb. 9	550	
Feb. 10	800	
Feb. 11	550	25
Feb. 12	775	
Feb. 13	625	12
Feb. 15	575	
Feb. 16	550	

Summary of Case 7: Constant leukopenia and granulopenia for two years. Six separate attacks of fever with marked prostration and necrotic ulcers in mouth, throat and rectum. Symptomatic but not hematological recovery in intervals. Treatment: X-ray exposure to long bones, bone marrow and liver feeding.

In addition to agranulocytosis with its unknown causation, reduction in the granular leukocytes may be produced by several well known factors. All such conditions may be convenient-

ly classified, therefore, under the general term of "the granulopenias," as follows:

#### THE GRANULOPENIAS

##### (A) Primary

1. Acute agranulocytosis (Schultz)
2. Chronic or recurrent agranulocytosis

##### (B) Secondary

1. Blood dyscrasias (Hem. diathesis)	Pernicious anemia Splenic anemia Acute leukemia Thrombocytopenia Aplastic anemia
2. Infectious	Sepsis Typhoid Influenza Malaria Exanthemata, etc.
a. Bacterial (Acute)	Focal infection
(Chronic)	
b. Toxic Diphtheria, etc.	
3. Chemical	
Benzene Arsenic, etc.	
4. Radiation	
Roentgen rays Radium	

As an example of a secondary granulopenia of the infectious type, the following is a case due to typhoid fever:

*Case 8.*—A white woman, aged sixty-four, was admitted to the hospital on August 22, 1928, complaining of pain and soreness in the anus and rectum, weakness, and fever. She had been under treatment for a rectal fistula for six weeks, and had been confined to bed for three weeks prior to admission with an acute febrile illness.

On admission, there were several small fistulae about the anus and buttocks with a zone of considerable inflammation around them. The abdomen was diffusely tender. The leukocyte count was 1,200 with a complete absence of polymorphonuclear cells. The hemoglobin was 79 per cent, red cell count 4,000,000. The Widal was negative. Van den Bergh was positive. The urine showed a trace of albumin.

The temperature rose rapidly to 105.8°, prostration was extreme, and death occurred on the third day after admission.

The autopsy findings were as follows: Moderate jaundice. Three small ulcers in the anus each 5 mm. in diameter. Heart, 250 gm., myocardium soft. Lungs, negative. Spleen, 200 gm., soft pulp. Liver, 1,775 gm., swollen. Numerous small punched out ulcers with necrotic bases in the terminal portion of the ileum, in the cecum, and in the transverse colon. Mesenteric and retroperitoneal nodes enlarged, soft and hyperplastic. Bone marrow, fatty. Blood culture positive for typhoid bacilli. Widal positive.

The following case also belongs in the group of secondary granulopenias of the infectious type, in this instance due to chronic sinusitis:

*Case 9.*—A white man, seventy-two years of age, began a succession of severe "colds" in April, 1931, which persisted up to the time of his admission to the Miller Hospital on June 19, 1931. Shortly before, he had noticed swelling in the region of the left eye with disturbed vision, particularly diplopia when trying to look up.

Examination revealed the right nostril occluded with a deflected septum, middle turbinate swollen and injected, and a scanty mucoid secretion but no pus. A few days later there was definite evidence of a subacute, suppurative spheno-ethmoiditis with a discharge of pus from that region. His blood count at that time was hemoglobin 65 per cent, erythrocytes 3,800,000, leukocytes, 1,500 with 32 per cent polymorphonuclears. This was essentially the same as on previous examinations in which the leukocyte count had varied from 2,050 to 4,800, with polymorphonuclears from 65 per cent down to 7 per cent. Following a transfusion a left middle turbinectomy and exploration of the ethmoidal and sphenoidal sinuses were carried out on June 28. A few days later, stiffness of the neck with a bilateral Kernig and clouding of the mentality developed. A purulent discharge continued from his sinuses and for more than a week his condition remained unchanged. He was then given two more transfusions which were followed by a distinct improvement.

Daily complete blood examinations until July 31 remained about the same with the hemoglobin varying from 54 to 76 per cent, leukocyte counts from 1,500 to 4,350, with polymorphonuclears from 10 to 60 per cent. Morphologically, the neutrophils showed toxic changes with many degenerative band forms, the monocytes exhibited toxic granulation and vacuolization, while some of the lymphocytes were atypical but not immature.

With clinical improvement the leukocytes increased, and from August 15 to October 9, rose from 4,550 to 8,800, with a polymorphonuclear range from 20 to 50 per cent. He was discharged recovered on October 9, 1931, with a hemoglobin of 71 per cent, erythrocytes 4,110,000, leukocytes 5,800, 48 per cent being neutrophils and 43 per cent lymphocytes.

#### SUMMARY AND CONCLUSIONS

Agranulocytosis presents three major characteristics: (1) the practically complete disappearance of the cells of the myeloid series from the bone marrow without demonstrable damage to the erythropoietic system or to the megakaryocytes; (2) a profound leukopenia with total or almost complete absence of granular leukocytes, and a lymphopenia, without hemorrhagic diathesis; (3) ulcerogangrenous lesions of the mucous surfaces, especially the mouth, throat, and anus. The disease shows a striking predilection for women of middle age or over, and is marked by a sudden onset, extreme prostration, high fever, and a rapid, frequently fatal course.

Although the exciting cause of agranulocytosis is unknown, leukopoietic deficiency may exist long before active clinical manifestations of the disease appear (Case 1).

We are of the opinion that in agranulocytosis there is primarily a selective, specific injury to the leukopoietic elements only of the bone marrow, and that in this limited sense the condition should be considered a distinct clinical entity.

We are further of the opinion that the attempts which have been made to depart from the original concept of the symptom-complex are misleading. Rather is it an advantage to preserve the syndrome as a distinct entity and to adhere, with minor modifications, to the cardinal diagnostic criteria originally laid down by Schultz in 1922.

A sharp distinction must be drawn between agranulocytosis, or primary granulopenia of unknown causation, on the one hand, and the granulopenias which are secondary to blood dyscrasias, infections, and chemical and radio-active agents, on the other. To facilitate this, a classification of the entire group of granulopenias is submitted.

The treatment of agranulocytosis consists of attempts to induce and maintain active leukocytic formation. Of the numerous measures employed to accomplish this through stimulation of the inactive bone marrow, the nucleotide preparations (pentnucleotide), which have recently been introduced, have proved to be the most effective. It must be recognized, however, that even with complete hematological recovery, mucosal ulcer-

tions may persist and eventually cause death (Case 6). Complete exhaustion of leukopoietic function predicates a fatal termination.

Nine cases are discussed in this report. The first five are typical examples of the acute fulminating type of agranulocytosis. The sixth case is of unusual interest in that despite complete hematological recovery, death occurred at the end of five months as a direct result of active mucosal ulcerations in the esophagus, trachea and bronchi. Case 7 represents a chronic recurrent form of agranulocytosis very rarely seen. In this instance a constant extreme leukopenia and a granulopenia had been known to exist for over two years, during which period six separate attacks of fever with marked prostration and necrotic ulcers in the mouth, throat and rectum had occurred, with symptomatic but not hematological recovery in the intervals. Cases 8 and 9 are examples of secondary granulopenias, Case 8 particularly illustrating the difficulties which may be encountered in the correct classification of a granulopenic syndrome.

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## ACUTE MONONUCLEOSIS OR GLANDULAR FEVER\*

### REPORT OF CASE

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I AM reporting this case chiefly for two reasons: first, because I believe these cases are more common than is generally recognized at present, and, second, to re-emphasize the importance of careful morphological studies of blood cells in all diseases or conditions involving any part of the hemopoietic system.

All of us have observed and recognized abnormal lymphoid reactions following certain infections. Turk,<sup>1</sup> Naegeli,<sup>10</sup> Cabot,<sup>3</sup> Cross<sup>4</sup> and many others have reported examples. The following question still remains unanswered, namely, why does apparently the same infection in one person cause a high degree of lymphoid reaction, in another a fatal leukemia; in one a lymphatic

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and another a myeloid reaction? One lives with full restitution, the other dies.

The case I am about to report is interesting in that it presents most of the typical clinical and

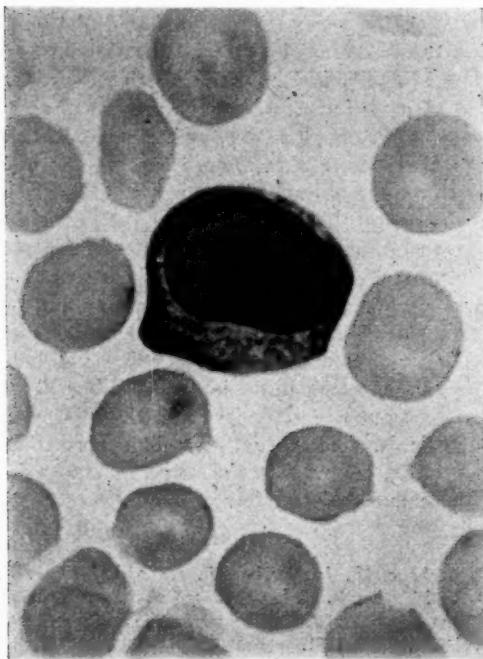


Fig. 1. Most common type of abnormal lymphocyte. Note coarse, hazy network of chromatin with some heavy strands and condensed masses not clearly separated from parachromatin.

#### hematological features of glandular fever and was first diagnosed as acute lymphatic leukemia.

The patient, L. K., is a young man twenty-two years of age who had always been well except for an attack of scarlet fever when fifteen years old. He came to my office on June 21, 1932, complaining of a fever of 100 to 103 degrees F., for the past two or three days. The week previous to that he had noticed he was very tired and had an almost constant headache. The week before that he had been on a fishing trip. Since having the fever, he had perspired freely at night, lacked strength or "pep" and had a constant dull pain over the lower abdomen not influenced by intake of food, action of bowels or bladder. The past two days he thought the glands in his neck were beginning to swell.

Physical examination at this time revealed a temperature of 101 F., pulse 84 and respirations 20. Eyes, ears and sinuses were normal. The throat and gums were very red and angry looking. Many of the cervical glands were enlarged and tender, as were also the axillary, intercostal and inguinal glands. The heart and lungs were normal and the blood pressure 90 systolic, 60 diastolic. Abdominal examination was negative ex-

cept for a slightly enlarged and palpable spleen. Urinalysis at this time was normal. A blood examination was as follows: Erythrocytes 5,230,000, hemoglobin 80 per cent, leukocytes 7,400. A differential count showed 45 per cent neutrophiles, 1 per cent basophiles, 2 per cent large monocytes, 50 per cent lymphocytes and 2 per cent plasma cells. A Mantoux intracutaneous test using 0.1 mg. O.T. proved to be negative. A blood test for syphilis, typhoid and Malta fever was also taken on the first visit, all of which proved to be negative. I advised him to go to bed, keep a record of temperature and pulse, and take a five grain amidopyrine tablet if his fever went over 103 or caused him discomfort.

The next day he felt much worse, his throat being more sore, and his headache and lower abdominal pain greater. The following two days he seemed about the same except that his temperature was gradually subsiding but the glands were enlarging. On June 25, his temperature was normal all day. He felt better but the glands all over his body, and the spleen, were much larger, the latter extending 2 to three cm. below the costal margin. His blood pressure was 100/65. The gums were red and spongy, bleeding on the slightest trauma. The throat was still very red and angry looking. A blood study on this day showed leukocytes to be 17,400, 25 per cent neutrophiles, 1 per cent eosinophiles, 14 per cent large monocytes and 60 per cent lymphocytes. His temperature remained normal from this time on, although he felt very weak. The pains in his head and lower abdomen gradually disappeared and the gums and throat became normal again in about a week. The enlargement of the glands and the spleen remained the same until July 6, a few new ones appearing each day in the neck, lastly in the submental region. From then on, the glands and spleen began slowly to diminish in size and seemed to reach normal about July 27. His strength and feeling of well-being returned about the same time.

The study of the blood picture during this time was exceedingly interesting. The first blood count made showed 7,400 leukocytes with 50 per cent lymphocytes and 2 per cent plasma cells. A morphological study of the cells showed many of these lymphocytes to be abnormal in appearance. The most common abnormal lymphocyte in my case corresponds closely to Type III, Figures 8, 9 and 10 in Downey's series<sup>5</sup> (Figs. 1, 2, and 3). The nucleus is often lobulated, and its size in relation to the cell varies. The chromatin forms a rather coarse hazy network with some heavy strands and condensed masses not clearly separated from the parachromatin (Fig. 1). In some of the cells, the arrangement of the chromatin is more diffuse, which gives it some semblance to a lymphoblast (Fig. 3). In others the chromatin is more coarse, resulting in a more definite network (Fig. 2). No definite nucleoli were found in any of the cells, although some showed small, dense, round masses of chromatin. Vacuolation was noted in some of the nuclei as well as the cytoplasm. Many of the cells showed very irregular nuclei, frequently placed eccentrically. The cytoplasm of most of the lymphocytes was the same, varying in amount with the size of the cell and varying in degree of basophilia.

With Wright's stain the spongioplasm was bluish-gray, finely granular on a somewhat yellowish background of hyaloplasm. Vacuoles were numerous and some of the cells contained azurophilic granules, fine and carmin red as usually seen in lymphocytes. This characteristic type

noticeable. The leukocytes numbered 7,800, with 68 per cent lymphocytes, most of which were still abnormal types.

On July 13 the count was 8,400, with 44 per cent lymphocytes; on July 20, 7,400, with 36 per cent lym-



Fig. 2. Chromatin is more coarse, resulting in a more definite network.

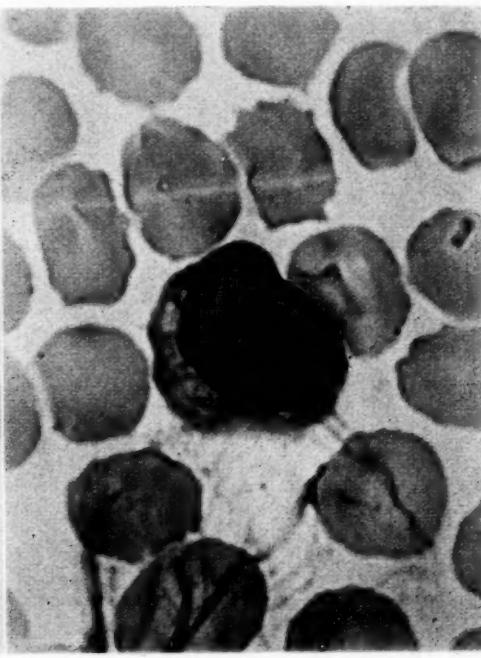


Fig. 3. Note diffuse arrangement of chromatin resembling a lymphoblast.

has been described by Downey as a "highly differentiated, mature leukocytoid lymphocyte." Occasionally a cell was seen with the nucleus smaller and the cytoplasm larger, not so basophilic but vacuolated. These resembled somewhat a plasm cell, derived from a lymphocyte (Fig. 4). They corresponded to Figure 7 of Downey's Type II.<sup>5</sup> Downey distinguished these types of abnormal lymphocytes and found that certain types predominated in certain cases. His Type III, illustrated by Figures 8, 9 and 10 in his plate, are similar to the predominating abnormal lymphocytes in my case. The polymorphonuclear leukocytes showed a shift to the left according to the Arneth classification. Many of the nuclei had only two or three lobes and were horseshoe in shape (Fig. 5).

The second leukocyte count, made on June 25, was 17,400, with 74 per cent lymphocytes, most of which were the abnormal types just described.

The next leukocyte count, made four days later, was 21,000, with 86 per cent lymphocytes. At this time his fever had disappeared but the glandular and splenic enlargement were at their peak.

He returned one week later, July 7, feeling much better. The adenopathy and splenomegaly were less

phocytes; on July 27, 8,800, with 26 per cent lymphocytes.

The spleen and lymph glands had returned to normal size by this last date and he was then feeling fine.

By August 10 the white cells numbered 7,800 with 31 per cent lymphocytes, most of which were still of the abnormal type. On September 22 the count was 9,900, lymphocytes 32 per cent, with about the same number of abnormal types of cells, though they were not so striking as in former slides. By November 11 the leukocyte count was 9,400, with 30 per cent lymphocytes. There were still many of the abnormal lymphocytes seen, but more of the normal type were present. He felt well and seemed to be in perfect health. No evidence of nephritis was found at any time.

One of the most interesting points to me about the blood picture in this case is that although the first leukocyte count was normal, the morphological picture of the blood cells was not. If they had not been studied, the diagnosis might have been missed or at least delayed. Again, four months later the clinical picture and the leukocyte count

had returned to normal, so-called atypical "leukocytoid" lymphocytes being still present in the blood. The diagnosis of glandular fever was made from the typical clinical picture, and characteristic blood findings.

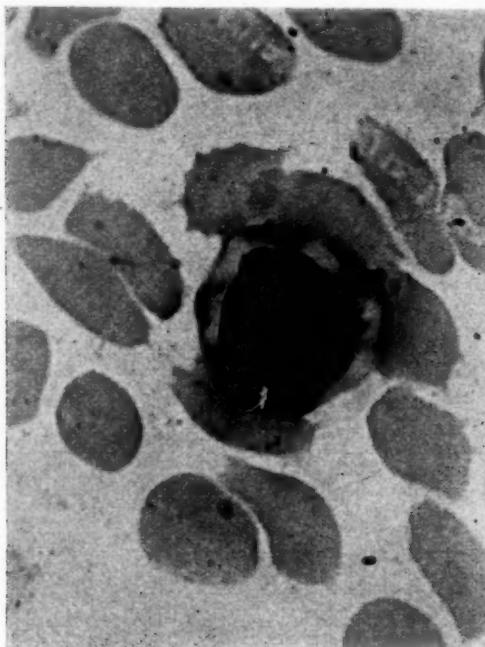


Fig. 4. This cell shows a smaller nucleus with more cytoplasm, the latter not so basophilic and containing many vacuoles. These cells resemble plasma cells, derived from a lymphocyte.

Pfeiffer,<sup>11</sup> occurring mostly in children, epidemic in nature, febrile, showing a red throat with non-tender cervical glandular enlargement, and frequent abdominal pain, enlarged liver and spleen.

The third group occurs more often in young



Fig. 5. A "leukocytoid" lymphocyte with two neutrophils showing two and three lobes to nuclei—Arneth's shift to left.

Glandular fever was first described by Pfeiffer<sup>1</sup> back in 1889, under the title of "Drusenfieber," but it was Tidy and Morley<sup>15</sup> (1921), Tidy and Daniel<sup>14</sup> (1921) and Downey and McKinlay<sup>5</sup> (1923), who established it as a clinical entity and accurately described the blood changes and clinical picture. It is evident from their writings that cases in the literature described as acute lymphadenosis, infective mononucleosis, acute benign leukemia, pseudo-leukemia and peculiar lymphoid reactions secondary to infection or sepsis are probably the same as glandular fever.

There are three groups or types of cases which have been reported. The first, one might describe as a miscellaneous group which seem to be peculiar or abnormal lymphatic reactions to infection or sepsis, such as reported by Turk,<sup>16</sup> Naegeli,<sup>10</sup> Herz,<sup>7</sup> Cross<sup>4</sup> and Cabot.<sup>3</sup> The second includes such cases as were first described by

adults and corresponds with those cases reported by Tidy and Daniel,<sup>14</sup> Sprunt and Evans,<sup>13</sup> Bloedorn and Houghton,<sup>2</sup> Downey and McKinlay,<sup>5</sup> Longcope,<sup>9</sup> and more recently by Benson<sup>1</sup> in England, Lehndorff and Schwarz<sup>8</sup> in Vienna, and others. In these, the glandular enlargement is more likely to be general with a preceding upper respiratory infection and systemic reaction. The spleen is usually enlarged. There are no hemorrhages nor secondary anemia. A leukocytosis with relative lymphocytosis is characteristic. Slight reddening of fauces and tonsils is usually present without exudation. The glands usually appear about the third day and reach a maximum in one to three days with an accompanying fever of 101 to 103 degrees. The spleen and glands usually subside in five to fifteen days, but may relapse or remain palpable for several weeks or months. Suppuration is rare. The fever usually

subsides within a week or two but has been known to persist longer. Recovery is usually complete within fifteen to thirty days except for the adenopathy and the abnormal blood picture which persist for months.

The disease has recently been prevalent on the continent, as indicated by the reports of Benson<sup>1</sup> in Britain, Rolleston<sup>12</sup> in Germany, and Lehndorff and Schwarz<sup>8</sup> in Austria.

Downey's and McKinlay's<sup>6</sup> article published in 1923 brought the subject up to date at that time and included a complete bibliography. The clinical phase was well presented by McKinlay and the hematological side thoroughly and masterfully dealt with by Downey, including an excellent plate illustrating the various types of pathological lymphocytes.

Lehndorff and Schwarz<sup>8</sup> have published a very extensive article entitled "Drusenfieber," covering over a hundred pages, including a discussion of the entire literature up to 1932. They feel that glandular fever is epidemic chiefly in children. An incubation period of seven to eight days is followed by a prodromal stage, usually quite short but which may last two to four weeks, during which the patient has general malaise, loss of appetite, abdominal pain, fatigue, etc. Then the fever begins and from this point they distinguish six types: septic, anginal, pharyngeal, thoracic, abdominal, and mild. (Time will not allow me to deal with them individually. Their names are explanatory.) The glandular enlargement is distributed more or less according to these types, the cervical adenitis being the most common.

They find that splenomegaly is one of the symptoms of this disease. Other symptoms recorded are bronchitis, rhinitis, pharyngitis, tonsillitis, abdominal pain and colic, liver enlargement with or without icterus, and exanthemata.

The chief complication is nephritis. All types from a simple albuminuria to a fatal hemorrhagic nephritis have been reported. The circulatory system is not particularly affected other than is usual in general infection. The nervous system has only rarely been involved, and then chiefly the cerebrum and meninges as reported by Epstein and Domeshek.<sup>9</sup> It is interesting to note that the increase in the cells of the spinal fluid was similar to those in the blood, namely, lymphocytic (34 cells—30 lymphocytes).

The etiology of the disease is still unknown. The presence of spirochetes in the throats of

many of the patients is not considered important or directly related.

Its differential diagnosis involves chiefly lymphatic leukemia from which it can always be distinguished by the morphological characteristics of the abnormal lymphocytes according to Downey.<sup>5</sup> The absence of anemia and purpura should help to distinguish it clinically. Tuberculosis, typhoid fever, Hodgkin's and other diseases involving the lymphatic system may simulate it at certain stages. Its complications may cause other diagnostic difficulties, namely, glomerular nephritis, encephalitis, etc.

*Conclusion.*—Matters of special importance for further study of glandular fever relate particularly to its etiology, diagnosis, and whether or not it is a definite disease entity, and, if so, with its mode of transmission, epidemiology and classification.

I acknowledge indebtedness to Dr. Hal Downey for his valuable advice in the study of the blood cells in this case.

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## GASTRIC AND DUODENAL ULCER: RESULTS OF IMMUNIZATION TREATMENT\*

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**A**S a prelude to the main theme of my paper I would like to present a brief résumé of the experimental investigation of elective localization of streptococci in ulcer, performed by Nickel and me in 1926 and 1927.

A series of gastric and of duodenal ulcers, and of gastrojejunal and other recurring ulcers was studied carefully from the standpoint of infection. Cultures were made of all available foci, and of the ulcers, if they were surgically resected. Part of each ulcer was kept for histopathologic study, as well as being stained for organisms by Rosenow's special technic. A green-producing, Gram-positive, diplostreptococcus was isolated in cultures from one or more foci of each patient with ulcer, except in those few instances in which foci had been completely eradicated previously. An organism of the same morphologic, cultural, and biologic characteristics was found in the resected ulcers, and could be demonstrated microscopically in the inflammatory zone surrounding the ulcer in all cases.

This diplococcus, isolated from either the focus or the ulcer of the patient, was capable of producing acute hemorrhagic erosions and shallow ulcers in 73 per cent of all animals inoculated, whereas streptococci isolated by Nickel in 138 cases of arthritis and inoculated in laboratory animals, produced lesions in the stomach or duodenum in only 5 per cent.

With the same organism foci of infection were artificially produced in devitalized teeth of healthy dogs, and resulted in the production in some of the animals of subacute and chronic forms of gastric ulcer in from one to six months. Likewise, the organism was demonstrated in

the ulcer microscopically and culturally, and was recovered from the foci when injected into other animals; the recovered organism had the same selective activity as it originally had. Strains capable of the greatest selective activity were kept for the production of a stock vaccine, designated ulcer vaccine, prepared in the usual manner, and containing 2,000,000,000 attenuated organisms in each cubic centimeter.

The vaccine of the ulcer-producing streptococcus seems to have more than a non-specific protein effect. Commercial catarrhal and typhoid vaccines given to a series of eight patients who entered the hospital, for whom diets and medication were not controlling symptoms, gave no relief with, or after, a series of four to six injections, whereas symptoms were promptly relieved after two or three injections of the ulcer vaccine.

Injections of aolan and a commercial lipin-proteid compound with emetine, and which is marketed as Synadol, gave relief of symptoms temporarily in some cases but apparently no immunity. A series of six patients treated by my hospital colleagues, with diet, alkali and Synadol, all received temporary relief of symptoms, probably due to temporary relief of pylorospasm and of hypermotility, as noted by roentgenologic examination, but there was no change in the ulcer, and in all cases symptoms recurred in less than a year.

I will admit that, in the last decade, volumes have been written on the cause and treatment of peptic ulcer and it may seem superfluous to try to add anything to the vast array of literature, but the problem is far from being solved. There is considerable diversity of opinion in the conclusions of different clinical and experimental in-

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vestigators. In most instances the hypotheses as to cause and proposed methods of treatment are directed at some one of the physiologic functions that has become perverted consequent to the ulcer itself. Therefore, treatment affords little more than temporary and symptomatic relief.

Two attributes of ulcer stand out: one, their tendency to heal, especially if measures are directed against hyperacidity and pylorospasm, and the other, their tendency to recur.

In consideration of treatment, therefore, all effort must be made to effect a change in the perverted physiologic processes, and in all factors of a predisposing nature, but of prime importance is removal of the basic cause of the ulcer.

Recent investigations have shown the importance of the presence of adequate amounts of mucin; this substance is now to be regarded as an important constituent of gastric content. All the chemical and mechanical factors which operate in the presence of ulcer are probably of secondary importance in the cause of ulcer. The constitutional and neurogenic factors so often referred to as important in the etiology are probably entirely predisposing.

In the treatment of peptic ulcer, since 1927, more emphasis has been placed on what has been suspected as being an exciting etiologic agent; namely, a specific diplostreptococcus. Also, more emphasis has been given to removal of the source of the infection, when possible, as well as on trying to build up immunity by the use of ulcer vaccine.

The series of 154 cases of ulcer which were seen in my private practice and which form the basis of this report was observed and treated in the period from May, 1927, to May, 1931, inclusive. All patients treated since May, 1931, were not included, because no generalization could be drawn from so short a period of observation. Cases seen in charity hospitals and similar clinics were not included. In all cases that were included, a definite diagnosis of peptic ulcer was made and confirmed by roentgenologic and laboratory evidence; the history also was definite, and therapeutic tests were included.

There were 137 men and seventeen women, or a ratio of about 8:1. The average age for the group was thirty-eight and eight-tenths years. The youngest patient was twenty years of age and the oldest sixty-seven years, and the average

duration of symptoms of ulcer was six and eight-tenths years. The history of shortest duration was one month, and that of longest duration, forty years. Ten patients had definite symptoms of ulcer of twenty or more years' duration.

The habits of living, considered by some as important contributory factors in production of ulcer, were carefully investigated, especially as regards irregularity of meals, and use of coffee, tobacco, and alcohol. In 35.7 per cent of cases all habits were good and in 64.3 per cent habits were considered bad. In a miscellaneous group of patients without ulcer, of corresponding age and ratio by sex, the habits of living of 58.2 per cent were bad. There was little difference between the habits of living of patients with ulcer and those without.

There was a history of influenza in 46.7 per cent of cases; of tonsillitis, in 22.4 per cent; of pneumonia, in 12.8 per cent; of appendicitis, in 12.8 per cent; of typhoid fever, in 7.4 per cent, and of rheumatic fever in 5.6 per cent. Two patients reported having had syphilitic infection, but it was not clinically active at the time of the investigation. One patient had tuberculosis, not active for many years, and one, acute catarrhal jaundice. In comparing an equal group of patients who did not have ulcer, the incidence of influenza was 48.1 per cent; of tonsillitis, 26.6 per cent; of pneumonia, 13.2 per cent; of appendicitis, 11.6 per cent; of typhoid fever, 11.4 per cent; of rheumatic fever, 2.8 per cent, and of syphilis, 3.5 per cent. Apparently such acute infections have no predisposing etiologic significance in ulcer but influenza is definitely known to aggravate symptoms of ulcer.

Only twelve of the 154 patients were free of clinical evidence that they harbored foci of infection on admission, but even they gave histories of removal of supposedly infected teeth or tonsils since onset of the symptoms of ulcer. Definite foci of infection were found in the tonsils in 71.9 per cent of the cases; in the teeth in 28.7 per cent; in the sinuses in 5.5 per cent, and in the prostate gland in 13.7 per cent. These percentages indicate that more than one of the structures concerned was infected in some cases. The incidence of foci of infection was ascertained among 120 patients of the same age and ratio by sex, who did not have ulcer, but who did have rheumatic arthritis, myositis, neuritis, recurring attacks of appendicitis or other diseases com-

monly attributed to focal infection; the tonsils were considered diseased in 53.4 per cent, the teeth in 26.7 per cent, the sinuses in 5.8 per cent and the prostate gland in 19.8 per cent.

Among the patients with duodenal ulcer, the average total acidity of gastric content was 60.2, and the average free acidity 45.3, in terms of cubic centimeters of normal sodium hydroxide. The highest total acidity was 98 and the highest free acidity, 74; the lowest total acidity was 25, and the lowest free acidity 15. The values for the patients with gastric ulcer were correspondingly lower. The average total acidity of gastric content was 51 and the average free acidity 38.8. The highest total acidity was 84 and the highest free acidity 69; the lowest total acidity was 15 and the lowest free acidity 5. The values for patients with gastrojejunal ulcer averaged, total acidity 43 and free acidity 31; the highest total acidity was 48 and the lowest free acidity 35; the lowest total acidity was 39 and the lowest free acidity 20.

In this group of 154 cases were 114 of duodenal ulcer, of which there was obstruction in thirteen and hemorrhage in three. There were also twenty cases of gastric ulcer, in two of which there was obstruction and in three hemorrhage. In thirteen cases of multiple ulcer, both gastric and duodenal, four of the ulcers were obstructing and three hemorrhagic. In seven cases of gastrojejunal ulcer, two were obstructing and one hemorrhagic.

The treatment was chiefly medical and consisted of a well-balanced bland diet, modified from the Sippy diet for ulcer that is recommended for the fourth week. The quantity and variety of food was gradually increased until the calories were sufficient for the patient to maintain weight and strength. Foods rich in vitamins A, B and C and in minerals were carefully retained in the diet. All patients were ambulatory except those markedly undernourished, or those with hemorrhage, obstruction, or slow perforation. The diet for the latter consisted of frequent regular feedings of liquid foods for the first few weeks. Alkaline powders, antispasmodic drugs and, when necessary, alkaline mineral oil and agar (Alkaline Petrolagar), were the only medications given orally. Instruction regarding hygiene, and a warning to avoid alcoholic beverages and tobacco, was given as a routine. When necessary, the stomach was aspirated nightly and

lavage was continued until secretions at night were controlled.

Four patients were operated on without any serious attempt at medical treatment other than that usually given before operation. Three other patients had to be operated on because of recurrence of the ulcer, and because it was refractory to further medical treatment.

A serious attempt was made to remove all evident foci of infection of patients treated either surgically or medically; furthermore, to all patients who could and would take the ulcer vaccine, subcutaneous injections of it were given twice weekly; the dosage was started with 0.1 c.c. and was increased from 0.1 to 0.2 c.c. each injection, until 1 c.c. was reached, and this was continued for eight to ten consecutive weeks. The value of inoculations of ulcer vaccine is apparent, I believe, in the following case.

A man, aged thirty-six years, had had symptoms of ulcer for approximately eight years, and in the last two years had frequent, and at times severe, hemorrhages. Roentgenograms revealed a definite duodenal ulcer of the niche type. The patient was treated by a competent physician, with regular Sippy diet, powders, antispasmodic drugs and rest in bed for varying periods of time; the tonsils and two abscessed teeth were removed. Because of inability to control the symptoms of ulcer and the acute attacks of pain that occurred in the abdomen at times, with severe hemorrhage, exploration was advised. At operation a definitely diseased appendix, a chronically infected gallbladder without stones, and a duodenal ulcer on the posterior wall of the cap were found. The surgeon deemed it more advisable to remove the diseased appendix and gallbladder and leave the ulcer, which he thought would now heal following medical treatment. For two months after the patient left the hospital there was no gastric distress. Then, in spite of careful diet, alkalinization, and rest in bed, the symptoms of ulcer recurred, and melena occurred about every ten days to two weeks. The patient was referred to me by his attending physician. Without any change in the previous treatment, inoculations of ulcer vaccine were given twice a week for ten weeks. All symptoms of ulcer rapidly subsided and no further hemorrhage occurred. Follow-up roentgenograms revealed that the niche in the duodenum had disappeared and there has been no recurrence of symptoms for the last three years.

#### RESULTS OF TREATMENT

In 1927 sixteen cases of ulcer were treated. In seven, treatment was by means of diet, alkaline powders, antispasmodic drugs, gastric aspiration and lavage. Two of the seven patients had all foci removed and one suffered recurrence of

symptoms of ulcer about a year later. The other five of the seven did not have foci removed and four of the five suffered recurrences in eight to eighteen months. Eight patients received, in addition to the usual medical regimen, inoculations with ulcer vaccine. Six of these had all foci removed, without recurrence of ulcer to date. Two of the eight did not have the foci removed, and the ulcer did not recur. One patient with a hemorrhagic ulcer was treated surgically and died following operation.

In 1928, thirty-one cases of ulcer were treated. Gastroenterostomy, without any serious attempt at medical treatment, was performed on one patient who had a bleeding gastric ulcer, and a definitely diseased appendix was removed; the ulcer has not recurred. Of the patients on the usual medical regimen, only four had foci removed and two of the four suffered recurrence of the ulcer. Three of the thirty-one did not have foci removed and ulcers of all recurred in eighteen months or less; of these, one patient was treated surgically, by gastro-enterostomy and removal of foci, without recurrence to date. Fourteen patients received ulcer vaccine and had foci removed, and in none have the ulcers recurred. Nine patients received ulcer vaccine, but as foci were not eradicated, one suffered a recurrence of ulcer two years later.

In 1929, forty-nine cases of ulcer were treated. One patient was operated on primarily because of a slowly perforating and obstructing duodenal ulcer; gastro-enterostomy was performed and a chronically diseased gallbladder was removed without recurrence of the ulcer. Of those patients for whom only the usual medical regimen was prescribed, eight had foci removed, and the ulcers of three of the eight recurred in one to two years. Seven patients of the forty-nine did not have foci removed and the ulcers of five of them recurred in ten to eighteen months. Twenty patients of the forty-nine received inoculations of ulcer vaccine and foci were eradicated. The ulcer of one patient of the twenty recurred and operation was performed one year following treatment; it recurred a second time and operation was performed again; the ulcer was resected and a posterior Polya type of resection was performed. The patient had a recurrence of the ulcer again this summer (1932). Thirteen patients were inoculated with ulcer vaccine but did

not have foci removed; the ulcers of three of the thirteen recurred two to two and a half years later.

In 1930, thirty-six cases of ulcer were treated. Two patients received only the usual medical regimen and had foci removed; the ulcer of one patient recurred at the end of eleven months. Gastro-enterostomy was performed on one patient with obstructing duodenal ulcer. The remaining thirty-three patients received, in addition, inoculations of ulcer vaccine; twenty-two of the thirty-three had all foci eradicated but ulcer of one patient recurred thirteen months after a severe attack of influenza. Eleven patients of the thirty-three did not have the foci eradicated and the ulcers of two recurred; one patient was operated on eighteen months after medical treatment.

In 1931, twenty-two cases of ulcer were treated. Of these patients who received only the usual medical regimen, two patients had foci removed and ulcer of one of the two recurred in one year. One patient of the twenty-two did not have foci eradicated, and ulcer recurred six months later. Of those who received inoculations with ulcer vaccine in addition to the usual medical care, nine patients had the foci removed and ulcer has not recurred. Ten patients did not have foci removed, and ulcer of two of the ten recurred ten and fifteen months later, respectively.

Of the entire group of 154 patients with ulcer in my private practice in the period of four years, 116 were treated with the stock ulcer vaccine in addition to diet and oral medication. Seventy-one of this group had all evident foci of infection removed, and ulcer of two only has recurred, an incidence of 2.8 per cent. Forty-five of the 116 patient received inoculations of ulcer vaccine but did not have all foci eradicated, and the ulcers of eight (17.8 per cent of forty-five) recurred in from one to three years. Thirty-four patients of the 154 did not receive inoculations of ulcer vaccine in addition to the usual regimen for ulcer but eighteen of the thirty-four had all evident foci of infection removed, and the ulcers of eight (44.4 per cent of the eighteen) have recurred to date. Among the sixteen patients of the thirty-four who did not have foci completely eradicated, the ulcers of thirteen (81 per cent of sixteen) recurred. Four patients of the 154 received surgical treatment only.

The clinical results in this series of cases of

ulcer seem to demonstrate that ulcer vaccine and removal of foci of infection are two indispensable adjuncts to the usual dietetic and hygienic measures, and offer more successful means of combating this chronic and recurring malady.

The series of cases is not large enough and the element of time is too short to draw any definite conclusions, but two deductions can be made: (1) that foci of infection seem to play an important part in the production of ulcer, and (2) that immunity, at least for a time, can be obtained by the use of vaccine made from streptococci obtained from ulcers of human beings, and having definite localizing properties when in-

oculated in animals. This would seem to substantiate the opinions long held by Rosenow.

Time and further effort will tell more, and fancy will give way to facts. There is still much to be learned from the careful correlation of clinical and laboratory data.

#### MEDICAL ARTS BUILDING.

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### USE OF MUCIN IN THE TREATMENT OF PEPTIC ULCER\*

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**A**LTHOUGH it has been demonstrated that mucin is of no definite value in the treatment of some cases of peptic ulcer, one should not be too ready to discourage its use. Undoubtedly mucin does have definite value in the treatment of some cases of benign ulcer of the stomach or duodenum, but it remains to be determined in which cases it can be used with the expectation of obtaining not only relief from symptoms but cure of the disease.

One of the important factors in most types of treatment of peptic ulcer, whether surgical or medical, seems to be reduction of gastric acidity. The more successful an operation is in accomplishing changes in the physiologic functions of the stomach, including reduction of acidity, the more successful it seems to be in permanently curing peptic ulcer. Gastro-enterostomy usually materially changes gastric chemical factors, and probably because of this it is frequently successful in accomplishing cure of peptic ulceration. In carrying out nonsurgical treatment, the effort

has been to accomplish reduction of gastric acids mainly by use of bland diet and alkalies. Objection to the use of alkalies has arisen because occasionally such use is followed by excessive secretory activity of the gastric glands. It is then difficult to control acidity satisfactorily, and the immoderate increase of alkalies to accomplish neutralization occasionally will result in toxic symptoms. Hardt and Rivers have shown that in 17 per cent of cases, following the Sippy regimen for treatment of peptic ulcer, signs of alkalemia developed. It is well known that certain patients tolerate alkalies poorly, particularly old people, and if there is any associated hepatic or renal disease; in such cases it may be extremely dangerous to advise the use of large amounts of alkalies. Recent experiments have led us to believe that among patients with advanced renal, and perhaps hepatic disease as well, it may be dangerous to use mucin indiscriminately.

There is still some uncertainty regarding the intimate mechanisms responsible for regulation of acidity of the stomach. Why the concentration of acid in the stomachs of certain persons seems to be persistently high, why in others the peak of acid secretion seems to be decidedly lower,

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whether the acid secreted by the gastric glands is poured forth in the same concentration under all conditions, or whether there is fluctuation in the concentration of the acid, remain undetermined; nor is it known exactly how significant the factor of duodenal regurgitation may be. It seems to be the consensus of opinion, however, that the mucus secreted by the gastric glands has something to do with regulation of acidity of the gastric content.

The mucous membrane of the stomach and duodenum, as well as of other portions of the alimentary tract, secretes mucus. It has been pointed out that local chemical and mechanical factors are largely responsible for its secretion. Excessive stimulation of nerves also will result in production of some mucus, although Fogelson and Ivy are of the opinion that this is of no great significance. Mucus in the stomach seems to have multiple functions, of which lubrication probably is one. Also, it may have some protective action for the gastric mucosa. Whitlow and Ivy suggested, "The mucus-containing cells of the gastric epithelium are the last layer of gastric wall to be digested by gastric juice . . . HCl-pepsin diffuses slowly through a film of mucus. . . . If the mucus film covering the mucosa of the pyloric antrum is wiped away, N/10 HCl application causes bleeding much sooner than otherwise." There seems, thus, to be some evidence that the adhesion of the viscid mucus to gastro-intestinal mucosa does protect, to a certain degree at least, these surfaces.

Control of gastric acidity, although possibly of minor significance, seems to be another of the numerous functions of gastric mucus. Mitchell suggested that mucus inhibits the proteolytic action of gastric juice "by a combined neutralizing, diluting, and buffering action." Heidenhain, in 1879, suggested that gastric mucus played some part in certain phases of neutralization of acid in the stomach. Pavlov, in 1910, maintained that the gastric mucus was responsible for certain fluctuations in acidity observed in the gastric secretion of pouches made from a dog's stomach. Kaufmann, in 1908, began using silver nitrate in the treatment of peptic ulcer, assuming that this substance produced an outflow of gastric mucus which favored the healing of ulcer. This method of treatment was soon discarded because of the development of numerous cases of argyria.

Recently Fogelson, on the assumption that gastric mucus was one of the substances which was intended by nature to regulate gastric acidity, suggested the feeding of mucus in order to lower the concentration of acid in gastric content. He prepared mucin from the mucosa of hogs' stomach and was able to show that this material would reduce acidity in the stomach of experimental animals. He felt that it might be applied in the treatment of peptic ulcer of human beings, and suggested that gastric mucus is an ideal antacid because: "(a) it combines readily with the free acid, (b) it is a natural substance which plays normally a protective, soothing, and lubricating rôle in the functioning of mucous membranes, and (c) its secretion or ingestion causes no chemical disturbances in the body and no unfavorable effect on gastro-intestinal secretory or motor activity." He reported the results of the use of mucin in a series of twelve cases of peptic ulcer, and maintained that there was definite improvement in symptoms. He further intimated that the relief from symptoms consequent to administration of mucin was due to two factors: "The mucin coated the ulcer and protected it against the proteolytic action of the gastric secretion, . . . through its high combining power with free acid it united with enough hydrochloric acid not only to neutralize the corrosive action of the gastric juice but to prolong the rate of dialysis of pepsin through the protective mucin layer."

In an unpublished article, Fogelson and Ivy reported further excellent results in the treatment of peptic ulcer with gastric mucin. They stated: "Gastric mucin controlled the symptoms in 101 out of 110 intractable ulcer patients. . . . No failure has been encountered in eighteen cases of acute massive hemorrhage treated with mucin. . . . In more than 450 peptic ulcer patients which included 110 intractable ulcer patients not responding to any other types of therapy, we were able to give complete subjective relief to over 90 per cent in from one to two weeks, with complete economic restoration." Atkinson, and Brown, Cromer, Jenkinson and Gilbert have brought forward additional evidence suggesting the efficacy of mucin in the treatment of peptic ulcer.

From Fogelson and Ivy's results it would appear that mucin has definite value in the treat-

ment of peptic ulcer, although the termination of symptoms must not make one too optimistic regarding permanent curative results.

We have used mucin in more than 150 cases of peptic ulcer. After several doses some of the patients refused it because of the objectionable taste or because it made them definitely more uncomfortable. A few of them were nauseated, and vomiting and diarrhea followed. In other cases symptoms were not controlled, and in such cases usually gastric acidity was not materially reduced.

We have recently reported on the discovery of a powerful secretagogue, probably histamine, in certain preparations of mucin. The product is now better standardized and seems to be practically free from this objectionable substance. It must not be assumed, however, that the commercial product which is now being marketed is pure mucin. Mucin is only one of its constituents. Liberal amounts of peptones are known to be present, and probably are chiefly responsible for the neutralization of acid. The various components of the product should be better understood before it can be used with confidence. We have recently noted elevations of the value for blood urea of certain patients with duodenal ulcer and associated renal disease who were given 60 to 80 gm. of mucin daily. Discontinuing the mucin would promptly be followed by return of urea to normal values. Whether these changes in the values for urea of certain patients were the result of the addition of very appreciable amounts of proteins included in the products of mucin now used, or whether they were caused by some specific toxin, has not been satisfactorily established as yet. It should also be stated here that among patients with duodenal ulcer and associated biliary disease at times signs of definite toxemia seem to develop, the nature of which is at present being investigated.

In certain cases mucin was used without particular regard to dietetic restrictions; in others it was used with a bland diet, and in still others in conjunction with small amounts of alkali. We soon found that it seems to be most beneficial when combined with other approved methods of treatment, such as a nonstimulating diet, sedatives, and a small amount of alkali.

It is our practice now to hospitalize patients during the periods when the ulcers are definitely active, and to place them on a regimen similar

to that instituted by Sippy, but using less alkali. Milk is given every hour. At 10:30 a. m., 4:30 p. m. and 7:30 p. m., 30 gm. of mucin is substituted for the usual hourly dose of alkali. The amount of alkali used varies, but seldom exceeds 120 grains (7 to 8 gm.) daily. After three or four days this regimen is modified; the patients are permitted to spend part of their time out of bed, and a more liberal diet, consisting of puréed vegetables, cereals, and various bland foods is prescribed. Instead of taking milk hourly they take it twice between meals. Alkalies are taken three times daily, 12 to 15 grains (0.8 to 1 gm.) of soda, bismuth, or magnesia one hour after meals, and mucin is taken an hour before meals and at 10:00 p. m., in doses of 20 to 25 gm. After three or four days on this diet an ambulatory regimen is substituted. The diet is limited to bland foods. Milk is given once between meals, and 10 or 15 grains (0.65 or 1 gm.) of alkali may be used an hour after each meal. Mucin is given an hour before the meal and before the patient retires; 60 to 70 gm. is used daily. Throughout the treatment patients are encouraged to get as much rest and relaxation as possible, and this is facilitated by means of liberal amounts of sedatives, such as bromides and the barbiturates. Usually atropine is combined with the other drugs.

Our dietitians have attempted to disguise the unpleasant taste of mucin by various combinations, none of which has been entirely successful. The method which seems fairly satisfactory is to dissolve 20 or 25 gm. of the powder in several ounces of warm water, stir thoroughly with an egg beater, and add 2 or 3 ounces (60 or 90 c.c.) of milk or malted milk flavored to suit the individual taste.

About 50 per cent of our patients responded favorably when mucin was employed in their treatment. We are therefore encouraged to continue its use, and we hope to ascertain, if possible, the types of cases in which good results may be expected. We believe, however, that in the presence of complications, such as obstruction, repeated gross hemorrhages, and evidence of malignancy or penetration, or if nonsurgical methods have proved of no avail, treatment will be more safely and satisfactorily carried out by operative measures. The remaining cases may arbitrarily be divided into two groups.

In the first group are cases in which local

changes in tissue seem of chief significance. As an illustration might be cited the case of a child aged five years who recently came under our observation. During an acute attack of tonsillitis, severe indigestion developed and large amounts of blood were passed by bowel. Roentgenograms revealed the presence of a duodenal ulcer. Infected material from the removed tonsils, when injected intravenously in experimental animals, produced hemorrhagic gastro-intestinal lesions. The use of mucin in this type of case, in conjunction with removal of foci of infection and other approved methods of treatment, would seem to be an ideal procedure in hastening healing. In this group, also, are acute ulcers, shallow mucosal erosions, gastritis, duodenitis, and gas-trojejunitis with or without associated ulcer, and chronic ulcer with intervals of exacerbation of symptoms which suggest an acute or subacute condition. In these cases there is a shifting of pain from the midepigastrium to the upper right abdominal quadrant, with local tissue reaction as evidenced by tenderness. Mucin might conceivably be of definite value in such cases, when local measures of protection are obviously desirable.

In the second group of cases systemic factors, especially those arising from a disturbed nervous system, seem of greatest significance. The patients are of the astute, intensive, high-strung type. During periods of fatigue, tension, and unusual worry, exacerbations of symptoms develop. These episodes arise with surprising rapidity following some unpleasant experience, and they may disappear with equal celerity. It is debatable whether such episodes always indicate activity of the ulcer, because similar types of patients under similar conditions not infrequently have similar syndromes, even though at operation ulcer is not demonstrable. Mechanical and chemical changes in the stomach, the result of nervous imbalance, may be responsible for the symptoms in these cases, and incidentally may be the factor which reactivates such ulcers at intervals. Giving mucin may be of value in these cases, but it seems reasonable that treatment directed toward systemic and especially nervous factors would be even more valuable. Readjustment of the activities of the patients, a

more sane program of living, more rest, more relaxation, large amounts of sedatives, and a bland diet would appear to be more essential than a substance which, after all, can be expected to exercise only a local action on gastroduodenal tissues.

#### CONCLUSIONS

Mucin, although probably of great benefit in the treatment of certain types of peptic ulcer, is not a panacea for the disease, nor is it in our opinion an adequate substitute for other approved methods of treatment. When attempting evaluation of its therapeutic worth it must be recalled that cessation of symptoms does not guarantee cure of ulcer.

The use of mucin for the treatment of peptic ulcer associated with disease of the urinary or biliary tract must be carried out with great caution because untoward effects may result from indiscriminate use of mucin in such cases.

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## MULTIPLE MYELOMA\*

PRECIPITATION OF BENCE-JONES PROTEIN IN THE BLOOD

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**M**ULTIPLE myeloma is not often seen in general practice, but because of the vagueness of the early symptoms, the lack of adequate laboratory and x-ray examinations, and the failure of the physician to suspect its presence when the patient has such vague complaints as lumbago, rheumatism, girdle pains, or neuralgia, a number of cases go undiagnosed, or the diagnosis is delayed until late manifestations of the disease make it obvious.

It is interesting to note that quite often the presence of multiple myeloma is suggested by some incidental laboratory finding, as the discovery of Bence-Jones protein in the urine (which occurs in about 50 per cent of the cases). For example, Short and Crawford report the precipitation of Bence-Jones protein in a patient's serum which was being heated at 56° C. in a Wassermann inactivating bath. This accidental finding resulted in the patient's being submitted to a complete x-ray examination, which showed the typical picture of multiple myeloma.

Recently, we had the opportunity to observe a case of multiple myeloma, and made some interesting observations on the blood chemistry of the patient.

### CASE REPORT

T. W. S., aged sixty-three, unmarried, was admitted to the hospital on September 5, 1932. His illness had begun the previous March, when he came down with a bad cold, had a non-productive cough, slight fever, and a general soreness over the front and sides of the chest. The pain in the chest was not influenced by deep respiration or by cough. During the next few weeks he felt better, but the soreness in his chest never left him. In May he began to complain of lumbago. He had dull aching sensations over the upper part of his spine, which were increased by exertion. He tried several home remedies, but these gave him no relief. During the summer, the soreness in his spine and in his chest slowly but progressively increased, and about a month before admission to the hospital he had begun to notice some tenderness in his scalp. There were no pains in the upper or lower extremities, and no loss of appetite. There was also no history of bleeding from

the nose, mouth, or bowel, and no history of urinary disturbances. Two weeks previously he had consulted his family physician and was told that the backache was due to kidney trouble.

*Physical examination.*—The patient was a well developed and well nourished elderly white male. There was not much evidence of loss in weight. His temperature was 97.8°; pulse 90. He appeared somewhat slow in his mental reactions and gave the impression of being drowsy and apathetic, although he was perfectly rational. A number of small irregular nodules could be felt under the scalp in the occipital and parietal regions. The pupils reacted normally to light and accommodation. There were small lumps present on the lower ribs. The lungs were normal. The heart was not enlarged and there were no murmurs. The blood pressure was 145/90. No tenderness, rigidity or spasm was found in the abdomen. No abdominal masses were palpable and the liver and spleen were not enlarged. The extremities showed no significant findings although the patellar, biceps, and triceps reflexes were a trifle sluggish. There were no disturbances of sensation. Rectal examination revealed moderate uniform enlargement of the prostate.

*Laboratory findings.*—Urine—specific gravity, 1.012; albumin, trace; many hyaline casts; occasional pus cell; Bence-Jones protein present. Blood—hemoglobin, 48 per cent; erythrocytes, 3,100,000; leukocytes, 8,400. Blood urea, 128 mgs. per 100 c.c. Blood Wassermann, negative; Kahn, negative.

During the course of the routine examination a determination of the blood urea was done, the mercury combining-power method described by Hench and Aldrich being used. In this method the blood is coagulated with trichloroacetic acid. When this was done and the mixture centrifuged, a fine milky white precipitate formed in the tube just above the layer of blood cells. This had never occurred in any of the previous tests we had done by this method, and we were unable to find any mention of it in the literature covering the test. The unknown precipitate was almost equal in volume to the layer of blood cells. To eliminate the possibility of faulty reagents as a cause for the unusual precipitation, the test was repeated with the same reagents on the blood or other patients, but no white precipitate formed. Personal communication with Dr. Hench informed us that this precipitate had not been previously observed by him.

The presence of Bence-Jones protein in the urine led us to suspect that this new precipitate might be Bence-Jones protein which had been precipitated from the patient's blood by the trichloroacetic acid. Oxalated whole blood was treated with trichloroacetic acid and

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centrifuged. The top layer of clear serum was removed, leaving the upper layer of the white precipitate and the lower layer of blood cells. The white precipitate was removed, washed with trichloracetic acid and again centrifuged. The acid was then decanted, and distilled water added to the precipitate. Heating caused



Fig. 1. X-ray of skull.

the precipitate to dissolve completely, but there was no return of cloudiness when the solution was cooled. To determine whether Bence-Jones protein was present in the clear solution two tests were done:

1. To 1 c.c. of the clear fluid were added three drops of sulphosalicylic acid. The solution became cloudy at room temperature, the cloudiness disappeared on boiling, and returned when the solution was cooled.

2. To five parts of the clear solution one part of 50 per cent acetic acid was added. This caused no visible reaction. To this, at room temperature, were added three parts of saturated solution of sodium chloride, which produced a cloudiness in the solution. The cloudiness cleared on boiling, and returned faintly when the solution was cooled.

We found, also, that the blood serum of this patient clouded when heated in a water bath at 56° C.

On x-ray examination of the thorax, spine, and skull, there were multiple spherical punched-out areas typical of multiple myeloma. The patient did not respond to x-ray therapy and died October 30, 1932. Autopsy confirmed the diagnosis of multiple myeloma.

The mercury combining-power method of determining blood urea, being a relatively simple method which can be done with a minimum expenditure of time and does not require an elaborate outlay of apparatus, is used frequently. Many physicians who do not have special laboratory facilities available use a commercial outfit in their offices. It is our belief that if the unusual phenomenon, described in this article, occurs when the test is being done, further examination should be made to discover the cause of this unusual manifestation, and the possibility of multiple myeloma should be borne in mind.

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#### SOME CLINICAL ASPECTS OF SPINAL INJURIES\*

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**FRACTURES** of the vertebra with or without dislocation are the most common results of severe injury to the spinal column. The vertebral column forms a bony protection for the spinal cord, which extends from the lower edge of the foramen magnum to the lower border of the body of the first lumbar vertebra and the whole range of spinal nerves are given off in this length so that in the intraspinal course of the nerve roots from above downwards there is an increasing

obliquity, greatest in the region of the cauda equina.

The most severe spinal injuries are those in which the spinal nerves or cord are injured. The spinal nerves are likely to be damaged in the intervertebral foramina and the cord in the vertebral canal. The canal is larger in the cervical, lumbar and sacral regions and smaller in the thoracic portion and it is this region in which spinal cord injuries are commonly severe.

The vertebra may be injured by direct violence,

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## CASE RECORDS

Case No.	Age	Sex	Etiology	Fracture	Dislocation	Paralysis	Therapy	Results	Post-mortem	
									Expectant	Improving
1	25	M	Struck by auto	None	None	Complete at level of 6th dorsal				
2	61	M	Struck by auto	Depressed skull fracture None of spine	None	Present	Elevation of skull None of spine	Died		Transverse myelitis 4th thoracic segment
3	54	M	Auto accident	Pedicle 4th cervical	3rd on 4th cervical	Present	Expectant			
4	36	M	Struck by auto	Spinous process 4th, 5th, 6th cervical	6th on 7th cervical	Present				
5	22	F	Auto accident	Spinous process 7th cervical Compound 7th and 9th dorsal	None	None	Expectant Stiff bed Taylor brace			Improved rapidly
6	19	M	Auto accident	Rami of 2nd cervical	2nd on 3rd cervical	None	Expectant			Developed pneumonia; recovered
7	44	M	Kicked in fight	Compression fracture 7th cervical	6th on 7th cervical Kyphosis	Present	Expectant			Transverse traumatic myelitis with crushing of 6th and 7th cervical
8	75	M	Struck by auto	Fracture of spine and lamina of atlas	None	None	Expectant			
9	26	M	Auto accident	Compression fracture 12th dorsal	None	None	Expectant Brace advised			
10	27	M	Fall 12 feet	1st lumbar	None	Present	Laminectomy elsewhere			Partial return, now stationary
11	7	F	Struck by auto	Spinous process 10th dorsal	10th on 11th dorsal	None	Hyperextension on frame			
12	46	M	Fall	Compression fracture 7th and 12th dorsal	None	None	Expectant			Improved
13	38	F	Fall 10 feet	Compression fracture of 12th dorsal Kyphosis	None	Present Transverse Myelitis	Bradford frame	Died		No autopsy
14	75	M	Fall 12 feet	Compound fracture 12th dorsal	None	None	Hyperextension Body cast			Improving is up and about

## SPINAL INJURIES—JONES

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					Present Transverse Myelitis	Splenectomy Laminectomy	Retrogressive Died 4-27-32	No autopsy
15	12	M	Bullet wound	Bullet passed to side of 12th dorsal	None	Hyperextension Body cast Taylor brace	Improved	
16	15	F	Toboggan accident	Compression frac. r.c. 4th lumbar	4th on 5th lumbar Posterior	Body cast	Improved Left under protest	
17	74	M	Fall 5 feet	Compression fracture 1st lumbar	None	Body cast	Improved	
18	50	M	Fall 15 feet	Compression com- minuted fracture 2nd lumbar Compression fracture 4th lumbar	3rd lumbar on 4th lumbar 4th laterally	Hyperextension Body cast	Improved	
19	26	M	Fall 15 feet	Compression fracture 2nd lumbar	None	Body cast Taylor brace	Improved	
20	51	M	Fall 25 feet	Compression fracture 2nd lumbar Kyphosis	None	Hyperextension Body cast	Improved	
21	55	M	Auto accident	Compression fracture 1st lumbar	None	Hyperextension Body cast	Improved	
22	16	F	Toboggan accident	Compression on fracture 1st lumbar	None	Hyperextension Body cast Taylor brace	Improved	
23	23	F	Toboggan accident	Compression com- minuted fracture 1st lumbar	None	Hyperextension	No improvement	
24	30	F	Auto accident	Compression com- minuted fracture 1st lumbar	None	Laminectomy 13 years ago	Some improvement Stationary for years	
25	55	F	Fall 3 feet	Compression fracture 1st lumbar	None	Taylor brace	Complete recovery	
26	17	F	Struck by auto	Compression fracture 2nd lumbar	None	Hyperextension Body cast Taylor brace	Improved	
27	45	M	Auto accident	Spinous process 5th, 6th, 7th cervical Compression fracture 7th cervical	None	Extension	Died 23 hours after accident	Transverse myelitis cervical cord
28	20	F	Auto accident	Compression fracture 7th dorsal	None	None	Improved	

causing fracture of the arches of the vertebra. They may be injured by indirect force, such as a fall on the head or buttocks, thus causing a crushing of the bodies of the vertebra. They may be injured by forced flexion or extension of the spine, causing a dislocation with or without an accompanying fracture of the bodies and articular processes of the vertebra. A dislocation without fracture may occur in the cervical region but is rare in other regions of the spine.

Damage to the spinal nerves or cord may result from the above lesions through laceration, contusion, hematoma or destruction from compression. The anatomical effect of such trauma is evidenced by the physical signs presented.

Complete interruption of the cord results in loss of function below the level of the injury. Many injuries cause incomplete functional involvement though at the onset it may appear to be complete. Patients suffering an incomplete lesion and affording some hope of recovery, often do not have a complete sensory loss. It is stated that in general the motor paths are more sensitive to injury and more easily show loss of function as a result of pressure. In the immediately complete lesions little hope can be held out for any recovery. When there has been an injury so severe as to cause destruction of the cord no regeneration is possible. This is not true, however, of the cauda equina as here peripheral regeneration is possible.

The nature of the injury may often be elicited by careful physical examination. In cervical dislocations the head is turned to the side opposite the injury. In compressed fractures and a majority of fracture dislocations there is angulation of the vertebral column with a prominence of the spinous processes. There may be an irregularity with lateral displacement of one spine on another. When evaluating the importance of these signs it must be borne in mind that particularly in the thoracic region irregularities of palpable parts of spinous processes may be normal. Great caution must be exercised when examining for a localized limitation of motion in case a fracture is suspected, as injury to the cord may be increased or actually produced by too thorough an investigation of movement.

A careful physical examination without radiographic aid often fails to detect a spinal injury without cord damage. The X-ray is of invaluable assistance in determining the extent of the

bony lesion in these cases. The studies must be carefully made and expert interpretation is of utmost importance.

In all cases in which there is any evidence of central nervous system involvement a competent neurological examination should be made and a very exact record kept of the findings. Future examinations can then be checked against these original findings and the progress of the case closely followed.

The term "broken back" always carries with it the impression that we are dealing with a very grave and often fatal condition. There are, however, many cases of severe spinal injury that do not have any involvement of the central nervous system and under proper management make a good recovery. It is also frequently observed that cases receiving a comparatively minor injury have spinal cord involvement. It seems that in the past few years there have been an increasing number of spinal injuries and this is undoubtedly due largely to the increased number of automobile accidents.

When there is the slightest suspicion of spinal injury a careful investigation should be made to ascertain the presence or absence of disturbance of the structure of the vertebral column. In the cases that have obvious involvement of the central nervous system one will usually find some accompanying bone lesion. Again a spine may appear normal upon thorough X-ray investigation and the patient be suffering from a complete transverse myelitis.

I wish to present a summary of twenty-eight cases, all but one of which came under my observation during the past three years and all but three of which were treated at the Ancker Hospital, Saint Paul.

The solution of any medical problem requires first a correct diagnosis and then there is little difficulty in carrying out suitable treatment.

There is considerable difference of opinion regarding the treatment of spinal injuries, especially if there is any damage to the nerve elements; but nearly all are agreed that given a case of spinal injury with evidence of immediate complete transverse myelitis, no surgical interference is indicated.

The fact that spinal injuries do not call for emergency surgery is of great importance. It is impossible to determine the extent of the injury to the nerve elements until the period of

## SUMMARY OF CASES

Etiology	Injuries	Treatment Received
Fall.....	Fracture..... 9	Operated..... 3
Auto Accident.....	Fracture and Dislocation..... 8	Hyper-extension Cast & Brace 14
Direct Violence (struck by car).....	Dislocation..... 7	Expectant..... 8
Toboggan Accident.....	No Fracture or Dislocation..... 3	Extensions..... 2
Bullet Wound.....	Paralysis..... 1	No Treatment..... 1
	Improvement..... 11	
	No Improvement..... 1	
	Died..... 5	

spinal shock has passed. This may last from a few hours to several days and operation undertaken during this period is attended by a high mortality.

Exploration is justified in cases in which one does not find complete paralysis but finds some evidence of conductivity remaining. In such a case a bony fragment may be found encroaching on the canal.

In compression fractures without paralysis, an attempt should be made to correct the deformity by hyperextension. After eight to twelve weeks the patient may be fitted with a proper brace and become ambulatory.

Unilateral dislocations without cord injury are susceptible to reduction. This is done under anesthesia by a combination of traction on the legs and head accompanied by gentle manipulation. Attempts at reduction should be made as soon as possible.

In cases when there is paralysis, especially of the bladder and rectum, very competent nursing is necessary and this should be closely supervised by the attending surgeon. Special attention must

be directed towards the care of the urinary bladder in order to avoid, as far as possible, the development of a severe cystitis. When catheterization must be resorted to the bladder should be irrigated with a saturated boric solution and this should be followed with instillation of a mild antiseptic solution.

Incontinence of feces requires mild laxatives and a cleansing enema daily.

All possible effort should be made to prevent decubitus. The position of the patient should be changed as frequently as possible. This avoids pressure being exerted too long on any particular area. The skin should be given careful attention through regular bathing and the application of alcohol and powders.

In conclusion I wish to emphasize three points:

1. Careful physical and X-ray examination in all cases of suspected injury to the vertebral column.
2. Spinal injuries are not surgical emergencies.
3. Operation is contra-indicated in cases suffering immediate complete transverse myelitis.

## THE HUMAN FOOT: FUNCTIONAL DEVELOPMENT AND WEAKNESSES\*

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DARWIN'S theory that man originated from the same ancestral stock as the great ape is generally accepted. The primates, the highest order of mammals, were the descendants of small, terrestrial quadrupeds who adopted an ar-

boreal life probably for protection. Their hind feet were characterized by long digits and short metatarsal bones with divergent halluces; feet suited for a secure, clutching grasp. As they developed the ability to run about in the trees, a keen sense of equilibrium was acquired, the creatures balancing themselves on the distal ends of the middle metatarsal bones. These bones be-

\*From the Section on Orthopedic Surgery, The Mayo Clinic, Rochester, Minnesota. Read before the Alumni Association of the University of Minnesota Medical School, Minneapolis, October 28, 1932.

came longer, the hallux lost some of its divergence, and the fourth and fifth digits became less prominent. The improved ability to maintain balance enabled them to squat when not in motion, a distinct advantage.

With the ability to range farther for food there came an increase in size of certain of these primates, and with it a basic alteration in the foot. The line of leverage of the foot became transferred from the third metatarsal bone and digit to a line between the first and second metatarsal bones. This latter line is called the "humanoid" line in contradistinction to the "primate" line, that extending through the third metatarsal bone. With its establishment the head of the second metatarsal bone became more important and extended beyond the head of the third.

As the centuries rolled on and the hind feet improved in their balancing and locomotive properties, the mode of locomotion changed. The hind feet were used chiefly as supports, and movement was accomplished mainly by swinging with the upper limbs, called by Sir Arthur Keith "brachiation." The semierect position seen in the modern anthropoid apes is probably due to the overdevelopment with consequent overweight of the upper extremities, due to brachiation. An important but little appreciated fact is that adoption of terrestrial life by our ancestors, after the erect posture had been acquired by arboreal habits, really was the fundamental change that made possible the development of human characteristics both mental and physical. The foot rendered this possible; its evolution is well worthy of study.

Terrestrial life caused the weight of the animal to be thrown entirely on the feet. With this change in function Wolff's law that "characteristic function and distinctive structure go hand in hand" came into play. Modification in structure of the foot was inevitable and the tracing of those changes is intensely interesting. The human foot probably began its distinctive development before the ancestral ape was of great size. The foot of this early anthropoid ape, as visioned by Morton, had the second metatarsal bone extending beyond the third, in accord with the humanoid line. The calcaneus was of moderate size, the foot was flexible, with but slight development of ligamentous structures, and was held in a slight degree of arboreal supination.

The metatarsal bones had marked torsion of their shafts, putting the hallux and digits into suitable position for arboreal grasping, as displayed by the modern apes. Morton, contrary to the opinion of many authorities, contends, with convincing arguments, that the supinated position had nothing to do with the development of that most important structure, the anatomic arch.

While our ancestors were still living in the trees, the foot was used for rapidly moving along the branches and limbs, and it was not called on for prolonged weight bearing when stationary, or for slow walking, with the characteristic heel and toe gait of modern man.

Function demanded of the modern foot has brought about a number of structural changes: the calcaneus and other tarsal bones have become broadened and strengthened; the great toe has advanced forward, and the first metatarsal has become enlarged; the insertion of certain muscles has been changed to give strength; the ligaments have increased in strength; the muscles of the calf have increased in size and strength, and the longitudinal arch has been gradually evolved and developed to its present importance. Failure in strength of the arch is the cause of most of the ills of the human foot. As an anatomic structure it varies greatly. In some persons with no complaint at all concerning the foot, the arch is very low, but, well developed or not, it must perform its function.

Two such authorities as Dudley J. Morton and Sir Arthur Keith differ decidedly on the relative importance of ligamentous and muscular support for the arch. The former insists that in the human foot the ligaments are the chief aid in furnishing a stable base on which body weight is to be supported, whereas the latter says that "a foot may be well balanced or ill balanced but we cannot, in either case, maintain the weight of our bodies posed on the soles of our feet unless every muscle of our legs and feet is in a state of reflex activity." It is probable that the ligaments are becoming of increasing importance. I have repeatedly seen patients, with the muscles of the foot and leg almost completely gone following infantile paralysis, still maintain a very good looking foot, with a good anatomic arch, performing well its function of weight bearing provided lateral displacements were prevented by the aid of braces. Both structures are of the greatest importance, but all of us are familiar with the

acutely painful, weak foot, the musculature of which seems without fault. The muscles most concerned in support of the arch are the two tibials and the two peroneals. The smaller muscles of the feet, the quadratus plantæ (flexor accessorius) and the interossei, are also of great importance. The quadratus plantæ in man arises from the calcaneus. It has two origins, one from the outer and the other from the inner surface of the calcaneus; the latter is seen only in man, and is inserted into the lateral margins of the tendon of the flexor digitorum longus. As the process of evolution continued, this muscle developed and strengthened the sole of the foot in no small way.

Before weight-bearing and progression could be carried out with the animal in the erect position it was necessary, as previously stated, that the tarsal bones increase in size and undergo modifications, and that the action and support accorded to the bony components of the foot by the various muscles be so altered as to permit of the foot maintaining the body weight for long periods. In the pronograde monkey (ape) the calcaneus is seen to be small and ill developed, and the plantaris muscle courses over its posterior extremity, to be inserted in the forepart of the foot. The heel does not touch the ground. In the orthograde gibbon (ape) the heel rests on the branch or ground and is seen to be better developed, and the tendon of the plantaris muscle has become divided at the calcaneus, so that the distal portion acts as the plantar fascia while the proximal portion of the tendon is inserted into the calcaneus. This muscle, authorities believe, is becoming of increasing importance to man and is developing in size and strength to aid the calf muscles, which provide the leverage so necessary for the modern heel and toe gait.

The tibialis anterior in the pronograde monkey is a double muscle, and acts from the foot on the leg. It does not prevent eversion of the foot; the "Y" ligament acts in that capacity. In the orthograde (gorilla) the muscle is single but still has two tendons. To help steady the foot the peroneus longus muscle comes around the outer side of the foot and is inserted to the base of the first metatarsal bone, and the flexor hallucis longus muscle maintains the great toe in apposition with the ground. Thus are established the rudiments of the longitudinal arch, and this is the first step in its evolution. The tibialis an-

terior muscle in man is inserted into the first cuneiform bone and the first metatarsal bone, giving support to the arch.

The tibialis posterior muscle in the gorilla, as in all primates except man, ends in the base of the second, third, and fourth metatarsal bones. Its attachment to the navicular and inner side of the foot is partial and secondary. In man its insertion has been transferred to the tuberosity of the navicular bone. By its tonic contraction it helps maintain the talonavicular joint in a state of equilibrium, and balances the leg, and weight of the body, over the heel.

The peroneus longus muscle has its fixed points of action from the upper two-thirds of the fibula, and from the base of the hallucial metatarsal bone. In man this latter attachment has broadened to include the first cuneiform and second metatarsal bone. If the weight of the body is properly balanced on the foot the peroneus longus muscle is in a state of postural contraction, counteracting and balancing its opponents, the tibial invertor muscles of the foot.

The peroneus brevis is the opponent morphologically of the tibialis posterior. No anatomic change has affected this muscle in the evolution of the higher primates; it balances the leg from the base of the fifth metatarsal bone.

So, with comparatively slight structural alteration, the four balancing muscles of the tarsus of the prehensile foot of our ancestors have been made to do duty in the foot of man. Because of postural activities, Sir Arthur Keith says the arch of the foot of man came into being, and by the healthy action of the four balancing muscles, the arch of the foot of man is safeguarded and maintained. The foot has developed from a prehensile, grasping organ into one that is purely supportive.

Bony changes consist, broadly, in a relative increase in the proportion of the tarsal elements, not in much change in the metatarsal elements, but in a considerable decrease in the phalangeal elements. This is as would be expected, for the erect posture has necessitated, for stability, a wider, firmer, and more posterior prolongation of the calcaneus, the weight being thrown on the back part of the foot instead of the front part. The relative length and importance of the first metatarsal bone with its digit steadily increased as it became a component of the arch.

The human foot is appreciated as a wonder-

ful organ when one considers the manner in which it accommodates itself to the ground on which it is called on to bear the weight of the body. With the increase in urban and easy living, with all its sequelæ, and the necessity of standing for long hours on cement, tile, or other unyielding surfaces, it is no wonder that considerable increase in ills of the foot has developed. Modern shoes tend to inhibit development of the important smaller muscles of the sole of the foot. Although foot ills are practically unknown among aborigines who go barefooted, or who wear flexible coverings such as the moccasin of our native Indians, nevertheless, the shoe is far superior to the moccasin for the unyielding surfaces that the foot of the urban dweller encounters.

If this interesting development of the bony, muscular and ligamentous structures of the foot and the simple mechanics of muscular support are borne in mind, one is better equipped to apply remedies for the common ailments due to foot-strain. I will consider, somewhat briefly, the two most common afflictions of the foot, weakness of the longitudinal arch and metatarsalgia, and their treatment. The relationship of arthritis to flat feet will not be considered as it will lead too far afield.

#### WEAKNESS OF LONGITUDINAL ARCH

The longitudinal arch is composed of a posterior pillar, the calcaneus and talus, and an anterior pillar, consisting of the rest of the foot in front of the ankle. The anterior pillar has two divisions: the inner one consists of the neck and head of the talus, the navicular bone, three cuneiform, and the inner three metatarsal bones; the outer division consists of the cuboid and outer two metatarsal bones. The arch is so often the cause of complaint that the term "fallen arches" has come to be used by the laity for all kinds of foot troubles. There may be no visible lowering of the arch and still a very painful foot exists.

I have arbitrarily classified the patients whom I wish to consider under four headings (Table I).

The first group consists of those with long, slender, elongated feet, relaxed particularly in the midtarsal region, pronated, moderately abducted, and with extremely flexible midtarsal and metatarsotarsal joints. This type of foot is thought

TABLE I. CLASSIFICATION OF PAINFUL FEET

1. Slender, elongated, with relaxed midtarsal region, pronated and abducted. Usually comparatively painless.
2. Weak, painful, no loss of arch, no eversion, acute flat foot. Likely to affect nurses, interns, and so forth.
3. Weak, painful, with eversion and lowering of the arch, semispastic, shortening of tendo achillis.
4. Weak, painful, with rigidity, eversion and pain on inversion. Patients are often obese adult males, floor-walkers, head waiters, train conductors, and so forth.

by some to be the result of a congenital tendency; undoubtedly it is often a familial characteristic. It looks weak, and is flat, but may be, and in many cases is, a foot that does its duty well, with no discomfort. In adolescence or before, children are often brought in by their parents because the foot is flat, but the patient has no complaint whatsoever. At this age it is probable that support by good, firm shoes, with proper fitting heels and a firm shank, do a great deal to strengthen the foot. No matter what is done in this way the foot remains flat, but excessive pronation and abduction may be prevented by correct shoes. These patients should be treated in childhood if possible, using firm shoes perhaps with a small, felt arch, raised inner side of sole and a small heel. The adult encounters difficulty in getting properly fitting shoes. They often accept too short a shoe to get one narrow enough, and callousness, and so forth, are the sequelæ. Treatment of this type of foot is most unsatisfactory, and if the patient wants permanent relief operative measures are necessary. Otherwise, they must be reconciled to some trouble. Arthrodesis of the navicular and first cuneiform bones is advised by Hoke, who argues logically that as the excessive flexibility in the cuneonavicular and the second and third intercuneiform joints interferes with the coördinated arch-lifting power of the tibialis posterior, tibialis anterior and flexor hallucis longus muscles, the arch must be maintained by fusion of certain of its component parts. He also lengthens the tendo achillis. Postoperative protection from any weight-bearing for at least eight weeks is essential, until bony fusion occurs. The result is a strong foot with the objectionable part of its flexibility done away with.

The second group is composed of patients with weak, painful feet, unaccompanied by appreciable

lowering of the longitudinal arch. This condition is often spoken of as acute flatfoot, and is seen not uncommonly among nurses, interns, and others, who are called on to be on their feet for long hours on hard, unyielding floors. The

for the shoes of these people for more than twenty years. When it is necessary to use an arch support, it is of felt and leather, and is fastened in the shoe. The patient should be instructed in the correct way of walking, with the



Fig. 1. Picking up marbles by means of the toes.



Fig. 2. Rolling a ball with the feet inverted.

incidence of this condition in training schools for nurses has been considerably lessened by careful inspection of the feet of all probationers, and seeing to it that they are provided with good, sensible shoes. Experience in Rochester since these examinations have been carried out in both training schools, has been that it is very rarely necessary to take a nurse off duty. It is far better for them to carry on if possible. The regular regimen under which the young probationer nurse lives may cause her weight to increase rapidly by 10 to 15 pounds. This, with the arduous duties on the floor, puts a great strain on the feet. The girl should be instructed not to be on her feet any more than is absolutely necessary, obtaining rest when off duty. Hot and cold foot baths, night and morning, followed by a brisk rub with a rough towel to increase circulation, and massage with alcohol or extract of Hamamelis tends to increase the vascular tone. Exercises, particularly those that will increase and enhance the power of the quadratus plantæ (accessorius) muscle and the flexor digitorum longus muscle, such as picking up marbles with the toes (Fig. 1), stretching of the peroneal muscles and those which go to make up the tendo achillis, and increasing the power of contraction of the tibialis anterior and tibialis posterior are beneficial (Fig. 2). I have not prescribed a metal arch support

feet straight ahead, and should learn the trick of flexing the toes when walking. This toe exercise will wear out more stockings but will increase the musculature of the sole of the foot. These persons should always be carefully examined to see that the pain in their feet is not due to an arthritic condition. Foci of infection, if present, must be eliminated. It is characteristic that in this type of painful feet there is little to be seen. There may be a little swelling, and localized tenderness along the arch in the region of the head of the astragalus.

The third group is composed of persons with weak, painful feet, with some eversion of the foot and lowering of the arch, with a semi-spastic condition of the peroneal muscles and with some shortening of the tendo achillis. The symptoms of patients in this group will be found to have been of longer duration than the symptoms of patients in the second group, and are accentuated. When the foot is grasped, and inversion is attempted, the patient will complain of pain over the outer side of the foot. Spasticity of the peroneal muscles will be noticed and perhaps spasticity of the tendo achillis. These people usually have run the gamut of arch supports, osteopathy, chiropractic, and so forth. Physiotherapy, consisting of application of heat and massage, and active and passive movements,

sometimes will bring relief. Too often, however, the patients do not respond to these efforts, and more drastic measures are necessary. As in the second group, arthritis must always be considered, and foci of infection eliminated. An occasional patient has the cold, clammy, flat feet that border on the arthritic type, and the condition has been called "vasospastic flat foot." The condition occurs in young persons, and the foot is often so painful that the patient is not able to work. Conservative measures do not give relief. Ganglionectomy of the lumbar sympathetic chain in a few cases has worked beautifully, but such a procedure is fit for only the very occasional case, and should be done only on clear-cut indications. Manipulation under anesthesia, thoroughly stretching the tendo achillis and peroneal muscles, and forcibly flexing the toes, breaking up any adhesions, followed by rest in a cast, with the foot held in inversion and dorsal flexion for about a week, is often successful. After the cast is taken off, active and passive motion must be carried out, and the shank of the shoe must be firm and strong. If arch supports are necessary they should be of felt and leather and not of steel. Steel arches require careful fitting and adjusting, and the foot has to adapt itself to the arch, not by any means always a painless process. If spasm of the peroneal muscles is extreme, division of their tendons is indicated. It is astonishing how soon the tendons will unite, and on account of this Sir Robert Jones many years ago insisted that a good sized piece of these tendons must actually be excised. Rest in a cast for a week should be followed by physiotherapy.

The fourth group is comprised of those patients with weak, flat, painful feet, with rigidity, eversion of the foot, and extreme sensitiveness on manipulation. Static, often combined with infectious, arthritis is associated in these cases. The patient walks along with no spring in his step, he avoids rough spots in the pavement and ground, and usually wears fairly heavy shoes with a firm sole. The condition is most often seen among men inclined to obesity, who are forty years of age or more. It is a most resistant type to treat, and if all the ordinary measures suggested have been tried and fail, there is nothing left except arthrodesis of the talocalcaneal, the calcaneocuboid and the cuneonavicular joints. Protection in a cast, with no weight-bearing, is necessary for from ten to twelve weeks after ar-

throdesis. It is seldom that patients are willing or able to give up so much time, and so they usually get along as best they can.

#### METATARSALGIA (MORTON'S TOE)

Metatarsalgia, or Morton's toe, has been recognized for years, but it is astonishing how often it is missed. It is a paroxysmal pain that is limited to the region of the fourth metatarsal bone and practically always comes on only when the patient is wearing shoes. It may be projected up along the foot and even up the limb. Some patients suffer severely and women often state that no matter where they are they must hurriedly take off their shoe when a paroxysm comes on and manipulate and squeeze the fore part of the foot. As a rule no abnormal physical signs are to be seen. The pain is often described by the patient as a burning, bursting pain in the toe. Many theories have been advanced to explain it, but none of the explanations satisfactorily fits all cases. The probabilities are that the wearing of shoes in some way throws the bones into such a position, particularly the fourth metatarsal, that they irritate the branches of the external plantar nerve and cause paroxysmal neuralgia. The condition never afflicts aborigines who go barefooted, and rarely do patients complain that they are afflicted at night, when their shoes are off. Amputation of the fourth toe and excision of the head of the fourth metatarsal bone, either alone or in conjunction with amputation of the fourth toe, has been done, but many years have passed since I have performed this operation. It has been possible to relieve patients either by fastening a transverse bar on the sole of the shoe, on the outside (Fig. 3), or by padding inside, so that the weight will be carried behind the head of the fourth metatarsal bone. The transverse bar is by all means, in my experience, the best device. All sorts of compromises, such as slipping a wedge of leather in the sole, and appliances of that sort, have not been nearly as satisfactory. All manner of apparatus and arch supports has been made for relief of this condition and if properly fitted, and worn in the proper position in the shoes, relief usually will be afforded. When the apparatus is first put in, constant supervision and adjusting are necessary, a provision often impossible to be carried out if patients come from a distance. It also requires considerable mechanical ingenuity and

skill, as well as willingness on the part of the surgeon and patient to fuss with it. Hence my preference for the transverse bar. Certain types of shoes, particularly those with an inflare for the front part of the foot, may be the cause, and



Fig. 3. Shoe with transverse bar in place on the sole.

the condition may leave on these shoes being abandoned.

#### SUMMARY

The foot of man, purely supportive, has been evolved from the relaxed, flat, grasping, prehensile foot of the ancestral primate ape. The

muscles were first used to grasp and steady the leg on the foot. As mode of locomotion changed and the erect posture was attained, the heel bone and tarsal bones became larger, the metatarsals and phalanges decreased in size, ligaments developed, particularly the plantar fascia, and the longitudinal arch came into being and heel and toe gait was acquired. The tibial, the peroneal and the flexor hallucis longus muscles and tendons were the most important, and later the intrinsic muscles of the sole, particularly the quadratus plantæ, aided. Inversion and eversion of the foot became the means whereby balance was maintained, and the strong muscles of the calf provided the leverage on the posterior portion of the calcaneus so essential in walking.

The common complaints referable to the foot are due to strain and giving way of the longitudinal arch. Four general groups of feet with reference to the longitudinal arch are recognizable: first, the flat, elongated, flexible, slender foot, often with no pain, pronated and everted; second, the acute, painful, weak foot of normal appearance usually the result of prolonged and unusual strain; third, a group in which the foot might be said to be an exaggeration of the foot in the second group, and fourth, the broken down, flat, rigid foot, usually seen in middle adult life. Treatment is briefly considered in the body of the paper.

Metatarsalgia is paroxysmal neuralgia affecting the branches of the external plantar nerve, causing distressing pain in the region of the fourth toe. It is due to wearing improper shoes, and relief almost always can be afforded by placing a leather bar transversely on the sole of the shoe so that the weight is carried just posterior to the heads of the metatarsal bones.

## PRESENT DAY CANCER PROBLEMS\*

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THE cancer problem rapidly is becoming one of the major problems of medical practice and hospital service. Practically all medical specialties are concerned with it, for no part of the body is immune to attack.

Cancer is one of the oldest known diseases. The Papyrus Ebers of 1500 B. C. mentions the disease, as does the oldest literature of India and Persia. Hippocrates describes cancer of the skin, breast, uterus and other internal organs. He differentiated between benign and malignant tumors and burned out cancer of the neck, the earliest recorded use of diathermy. Preceding Hippocrates, Democedes, in 520 B. C., cured Atossa, wife of Darius Hystaspis, of cancer of the breast. The writings of Galen, Cato, Pliny and Celsus, to mention but a few of the men known to earliest medicine, contain frequent references to malignancy and its treatment.

Since 1927 cancer has held second place as a cause of death in the United States, being exceeded only by heart disease. The latest statistics from the Bureau of the Census, those for 1930, show that 115,265 deaths were recorded from malignancy, giving a death rate of 97.2 per 100,000 during that year, the highest ever recorded for this disease in this country. It may be of interest to note that in 1900 tuberculosis stood first as a cause of death with a rate of more than 200 per 100,000, and cancer was in sixth place with a rate of 63 per 100,000. In 1930, tuberculosis had dropped to sixth place with a rate of 71.5, while cancer had advanced to second place with a rate of 97.2. In Minnesota cancer deaths are increasing at the rate of approximately 100 each year, and now amount to one in eight, or 12.5 per cent, of all deaths within the state. There were 3,199 cancer deaths in Minnesota last year, or one every two and three-fourths hours.

Education has had much to do with the rapidly declining death rate from tuberculosis, and it is not too much to expect that cancer will in due time respond to similar measures.

The question is frequently asked whether cancer is increasing, and much ink is spilled in arguing the pros and cons. It is true that a larger percentage of the population is living in the cancer age period and that diagnostic methods are more refined than formerly. Whether these two important factors are the answer to this question is not so important as is the fact that more people are dying of cancer each year, and the major problem is not so much statistical as it is the finding of ways and means for reducing this rapidly mounting death rate.

It is only within the past thirty years that any particular attention has been given to cancer from the standpoint of research into its clinical and etiological factors. Up to that time the disease was first seen so late in its course that little, if anything, could be done for it. Radium and deep therapy were unknown in the treatment of this disease and surgery was the only form of therapy used. Even today, irradiation is viewed askance by some surgeons, although it is now definitely accepted as the method of choice in treating cancer of the cervix, skin, and buccal cavity.

Since 1900 marked progress has been made in the diagnosis and treatment of malignancy. Certain fairly definite indications of early cancer and pre-cancerous lesions are now recognized and accepted by workers in this field. As examples I might mention unrepaired cervical lacerations following childbirth; the combination of a luetic history, leukoplakia, neglected mouth hygiene and the use of tobacco; the presence of non-hairy, dark colored moles on the body surface; the keratotic, scaly, moist patch on the skin of the face and the scalp, as fair indications of the development of cancer in the parts mentioned. Very recently, Schiller of Vienna has described a test for cancer of the cervix in its preclinical stage. By painting the cervix and adnexa with Lugol's solution early cancer will be indicated by lack of staining in the area. Biopsy in that area will show beginning malignant degeneration.

Transillumination of the breast, developed by

\*Read before the Minneapolis Surgical Society, November 3, 1932.

Dr. Max Cutler of Michael Reese Hospital, Chicago, while not as selective as the method just described, gives valuable information regarding the character of the tumor mass in the mammary gland. By this simple means, which can be carried out at little added expense, clear fluid cysts, solid tumors, hematoma, papilloma and mazoplasia or chronic mastitis can be differentiated as well as definitely localized in the mammary gland. This method also has value in proving to a woman concerned about a possible breast malignancy that in the absence of such a malignancy the breast will show a uniformly translucent appearance which she can see for herself.

The x-ray is of incalculable value in the diagnosis of bone and intra-abdominal malignancy and obviates the necessity of surgical exploration in many cases.

The value of biopsies in the diagnosis of cancer needs no special pleading before this audience. Its utility and value when properly done carry little risk to the patient and it gives the most accurate answer to the type of tumor.

These few illustrations will serve to indicate some of the marked advances in the diagnosis and treatment of malignancy that have been made in the past few years, certainly within the professional experience of many in this audience.

Aside from the delay by the laity to seek early medical care, which will be discussed a little later, some of the retarding factors to an improved cancer service have been the inability of some physicians to appreciate the possibility of malignancy or to diagnose cancer when the patient first presents himself for examination, followed by a period of "watchful waiting" for more positive diagnostic signs, a period always fraught with the utmost danger to the patient as each day's delay lessens the chance for a cure. Another class of physician has been charged with temporizing with his treatment in order to increase his fees in the case. This type of physician we know to be in the vast minority, but like the proverbial rotten apple that spoils the barrel of good fruit, one such individual can cast a shadow over the ability and integrity of all other physicians in the community in which he resides.

The use of deep x-ray therapy and radium as therapeutic agents in the treatment of malignancy has been definitely established, yet in many communities their utility is questioned by some physicians, principally due to the unfavorable results

seen. These results have been brought about by the abuse of these agents at the hands of untrained physicians rather than by their use by competent men. When properly used they have a distinct place in the therapeutic armamentarium of malignancy, but they are also the most highly specialized forms of therapy known to medical science. To be used safely and effectively, the physician must know not only the type of tumor he is treating, that is, whether it is radiosensitive or radioresistant, but also the proper dosage, methods of implantation, proper filtration and screening, tissue penetration and tissue reaction, to say nothing of the problems of electricity and physics involved.

In general, the practice of supplying radium and radium emanation to the average physician for use in the treatment of malignancy will add little to a solution of the problem or to the benefit of the patient. Seldom will the proper dosage be given. Either the patient will be undertreated in order to avoid tissue damage and damage suits, thereby converting an originally sensitive tumor into a resistant one making any subsequent irradiation difficult, or the patient will be overtreated and permanently damaged.

The Report of the Royal Commission of Ontario on the Use of Radium and X-rays in the Treatment of the Sick, just published, says on page 100:

"Successful results in the use of these remedies depend upon their use in skilled hands and upon early diagnosis. Inadequate or unskilled first treatment by radiotherapy is liable to hasten the progress of the disease."

Further in their recommendations, on page 106, it is stated:

"Your Commission advises that no center be established until competent personnel for such center is available and in this connection desires to emphasize the fact that in unskilled hands such potent weapons as x-rays and radium may do more harm than good."

The pessimism that obtains in the minds of many physicians regarding the possibility of cure of malignancy should be replaced by a more optimistic attitude. It is now known that while a complete recovery cannot be expected in many cases, a prolonged period of comfortable living can be given these patients by proper palliation. Frequently such patients will violate all prognoses and become permanently cured.

At the recent St. Louis meeting of the American College of Surgeons more than 8,800 five-year cures of cancer were reported. Doubtless there are many thousand more such cases. Cancer is curable, and is one of the few diseases in the catalogue of medicine that will respond entirely to the skill of the physician. It is not a self-limiting disease as are practically all other diseases, but must be eradicated completely to save the patient's life, and in this eradication the skill of the physician is the all-important factor.

In addition to providing adequate facilities for the diagnosis and treatment of cancer, the major responsibility of the hospital, which it must share with the physician in the case, is that of adequate records and follow-up on cancer patients. No cancer case should be considered closed until the death certificate is added to the record. Freedom from the disease for twenty years has been followed by fatal recurrences. Periodic contact should be maintained with all cancer patients for as long a time as possible, preferably for life. This is often difficult for the physician or hospital alone, but when a joint undertaking is launched, especially when trained medical social workers are available, few, if any, cases will be lost. Some hospitals report 100 per cent success in their follow-up, while others rank lower in this regard. In any event a large percentage of post-treatment contacts can be maintained when there is full coöperation between the hospital and its staff.

The major problem with the laity is that of getting patients to seek medical care as soon as they suspect or know something is wrong. In spite of all the educational work that has been done an average of more than six months' time still elapses after the patient suspects or knows something is wrong and seeks medical aid. One reason for this delay is that there is little discomfort or pain associated with most forms of malignancy in early stages. There are also two outstanding fallacies in the public mind regarding cancer; one, that the disease is always fatal regardless of how early it is diagnosed; the other, that there is a social disgrace connected with this disease, therefore it is concealed as long as possible. Both ideas are erroneous and greatly increase the problems of prevention and control.

Some physicians criticise cancer educational work among the laity because of the fears or phobia created in the minds of a few neurotic individuals. A rather careful investigation of

this problem among a large group of applicants for admission to cancer hospitals and clinics has shown the fallacy of this situation, as but 2 per cent of 2,000 admissions could be placed in this class. These were neurotic individuals who, in the absence of a cancerphobia, would entertain a phobia of falling hair or fallen arches or anything in between. Instead of a fear of dying of cancer there should be encouraged a fear of beginning cancer and a fear of delay in seeking treatment. The public should be led to know that cancer is curable, with the odds almost entirely in favor of the early case. No one is better fitted to give this information to the public than the physician.

The question of hiding from the patient the true nature of his disease is still a debatable one. More and more physicians and hospitals are adopting a method of frankness in this matter and I believe it to be a sane and practical method to follow. The time is past when the medical profession can accomplish much good by being secretive in such matters. The public is fed daily by the press and the radio information on disease and health subjects. Few people remain long in ignorance about such matters. Probably more physicians are fooled by their patients who do not let on that they know they have the disease than are able to keep their patients in blissful ignorance of their true condition. Frankness invites coöperation, while sidestepping the issue often raises doubts in the patient's mind about his true condition and also about the ability and integrity of the physician in charge of the case. Lack of frankness often drives patients into the hands of quacks who are too brutally frank, and also tends to make "medical shoppers" of patients seeking the answer to their problem. Physicians and hospitals who adopt a frank attitude on this question report the fullest coöperation from their patients.

This very brief outline of some of the major factors in the malignancy problem suggests that the solution is neither a simple nor easy one. No standardized methods of procedure have been or can be applied because of varying local conditions. It is felt that none should be attempted until the facts bearing on each community are known and they can be determined only by careful investigation.

Such surveys as have been made have brought out interesting facts. It has been found that approximately 2 per cent of the average general

hospital adult intake is for cancer. About 25 per cent of the probable cancer patients are hospitalized during a given year. It is estimated that there are three living cases for each death during a year, and strangely enough statistics show us that the number of deaths from cancer in a given state during a year closely approximates the number of licensed physicians in that state at that time. Many times when this comparison is made on a county basis the figures are identical. It is also found on the above basis that there would be but three cancer patients per physician were all distributed equally among the profession.

Where facts of this nature have been developed, the problem has been clarified materially. Relate them to the hospital facilities for the diagnosis and treatment of this disease and the problem for any community is fairly well established.

The lack of facilities in many communities, such as those for adequate laboratory diagnosis or for treatment by means other than surgery, indicates the desirability of reference of cancer patients to institutions where adequate service can be had; either this or the development of adequate facilities within the community. Because of the small number of cancer patients seen in the smaller general hospitals it is not practical or logical to expect that facilities for complete care of these patients will be provided in such institutions. Present methods of treating tuberculosis suggest the possibility of providing centers for the care of cancer patients where they will be under the care of a staff specially trained in this work.

To render adequate cancer service today a hospital should provide facilities for the histological examination of tumor tissue by frozen section as well as by paraffin sections and pathologists trained in this field and competent to interpret the microscopic picture; competent surgeons; deep therapy with a minimum effective capacity of 200,000 volts frequently calibrated and with a competent roentgenologist in charge, and 150 milligrams or more of radium in a variety of containers, this also to be in charge of a trained radium therapist. Facilities should also be available for record keeping and follow-up on all patients using this service. When facilities are lacking in part there should be no hesitancy in referring patients to other institutions where such facilities are available. The welfare of the patient will be best served and the reputation of the

physician enhanced by such reference. In every instance, treatment should be predicated on laboratory findings as to the nature of the tumor.

Cancer is not a "one man" disease. As just indicated, the pathologist, the roentgenologist, the radium therapist, and generally the internist in addition to the surgeon or surgical specialist in whose field the tumor lies, has a responsibility to the patient. Their opinions are of value in arriving at the diagnosis and treatment of each and every case.

Time will not permit me to discuss this point further, and I shall refer you to the minimum standards for organized cancer service in general hospitals promulgated by the American College of Surgeons for an elaboration of this procedure.

When adequate facilities for the diagnosis and treatment of malignancy are provided and competent personnel is available in a number of centers in each state, it will then be in order to institute intensive lay educational campaigns to bring about early diagnosis. Too often at the present time facilities are lacking in many places properly to care for the cancer patient, and it is believed that the best interests of all will be conserved by concentrating on the development of adequate professional and hospital facilities for the diagnosis and treatment of this disease.

Cancer is a problem for the medical profession and the hospitals, and the interest of the intelligent public should be restricted to strengthening the resources of these two groups. How long lay activities can be held to this purpose depends on the wisdom and alacrity with which the profession and the hospitals meet the situation. Should undue delay or failure result it can only be expected that public demand will be met by state participation in the program. That this possibility is more than a mere fantasy may be gathered from the fact that nine states now make cancer a reportable disease, and in one other state, Massachusetts, with a state-wide cancer program under the Department of Health, the law provides that this work shall be done "with or without" the coöperation of the local physicians.

One other resource or agency should be added to those mentioned as having an interest and responsibility in this work. I refer to the official health department. Inasmuch as one of the chief functions of such departments is that of health education they have a distinct place in the cancer

control program. Official health agencies should not enter the diagnostic or therapeutic field but should confine themselves to educational activities. Where adequate laboratory facilities are not available or cannot be made available otherwise for the examination and interpretation of tumor tissue, the health department might meet this need through an expansion of its laboratory service, thus providing a service for the physicians of their jurisdiction. Also such departments can render useful service in the follow-up of cancer patients through their public health nurses and vital statistics bureaus.

Before concluding I would like to say a word about the American Society for the Control of Cancer and its relation to the problems discussed above. The object of the Society, as stated in its certificate of incorporation, is "to collect, collate and disseminate information concerning the symptoms, diagnosis, treatment and prevention of cancer; to investigate the conditions under which cancer is found and to compile statistics in regard thereto." These objectives have been interpreted broadly as educational in character. After spending several years in various forms of lay educational work, the Society has concentrated its efforts on working co-operatively with the medical profession and the hospitals in the improvement of facilities for the better care of cancer patients. It is in keeping with this purpose that the survey now under way in Minnesota was undertaken. Having determined on this policy the Society looks to the regular medical organization of the state and the various counties as the representatives of the profession in these areas and considers them the basis of its activities in a given community. For instance, no activities beyond those of a most general nature are undertaken in a community until an invitation is received from the medical society to do so, and reports on activities undertaken as a result of such invitations are made to the medical society issuing the invitation. The Society distributes literature similar to that available this evening. It also furnishes without cost except

return transportation charges the film shown at this meeting. It strives in any and all ways to be of service to all responsible groups interested in cancer prevention and control.

The state and local branches of the American Society for the Control of Cancer are organized on a plan similar to that of the medical societies. There are state committees and local committees. The state committee is under a state chairman appointed by the Executive Committee of the Society. He is a physician in good standing in his state medical society and able to work with the different interests involved in a given community. The state committee is composed of both professional and lay members who because of their interest are willing and able to contribute to the promotion of the work. Where sufficient interest is shown in a local community a local committee, with a physician as chairman and composed as is the state committee, is formed to work in that area. These local committees are expected to work with and through the state committee and state chairman and confine their activities to their local territory unless invited by the state chairman to participate in a larger program.

The purpose of these committees is to help the medical profession and the hospitals in all practical ways to render a better service to cancer patients in their respective communities, and when conditions warrant assist in lay educational activities.

It is believed that with the education of the medical profession accepted by the medical organizations as their responsibility and with the official health agencies providing health educational facilities to the public, and with the state and local committees of the American Society for the Control of Cancer assisting both groups to do the best work possible, a united and constructive attack can be made on the cancer problem that in time will be reflected in the correction of many precancerous conditions before they have developed into frank malignancy, and in reduction of mortality from this disease.

## UNDULANT FEVER TREATED WITH METAPHEN\*

A. C. FORTNEY, M.D.  
Fargo, North Dakota

MANY drugs and therapeutic measures have been advanced for the treatment of undulant fever, but most of them have been discarded as unsatisfactory. Since the disease, at least in its early stages, is a bacteriemia, any drug that, injected into the blood stream, will kill the organisms, with a minimal amount of reaction, is highly desirable. My attention was first attracted to metaphen by various reports in the *Journal of American Medical Association*, showing its high bactericidal power with low incidence of reactions and complications. I have used the drug in other blood stream infections with gratifying results, and so decided to use it for undulant fever, at the first opportunity.

### CASE REPORT

G. M., a white female, aged forty-one, housewife, entered the Clinic on January 12, 1933, complaining of a severe frontal headache, which had begun three months before. It had been getting worse and at the time of admittance was persistent night and day. Nothing important was revealed in her history except that two weeks before she had had a severe head cold with a rather profuse nasal discharge, which cleared up in a week. Her first physical examination revealed only bilateral clouded maxillary sinuses. She was referred to Dr. G. A. Larson, who washed out considerable pus from both antra. Her headaches were promptly relieved. Examination on her first visit showed: temperature 99; w.b.c. 6,150; r.b.c. 4,660,000; hemoglobin 80; urine normal.

On January 16, I was called to the house because she was having chills and fever. On examination her temperature was found to be 101 F. and she was having chills followed by drenching sweats, every eight hours, with clock-like regularity. At the time of the chill her temperature was usually found to be 103 or 104. Three or four hours later the temperature would be 98. When her temperature was normal, the patient felt perfectly comfortable and certainly did not appear as ill as might have been expected for one who just a few hours before had had a temperature of 103 or 104. Now her white blood count was found to be 4,600 with 40 per cent lymphocytes, 30 per cent segmented, 19 per cent staff, 2 per cent eosinophiles, and 9 per cent monocytes. Undulant fever was suspected, particularly as it was found that she had been drinking raw milk from a

neighboring farm. However, the first specimen of blood was returned negative for undulant, typhoid and paratyphoid fevers.

The above picture continued for three days and then the patient was hospitalized. A second specimen of blood was then examined and on January 19, 1933, agglutination for undulant fever was found positive in dilutions of 1:640. Ten c.c. of metaphen 1:1000 was then given intravenously, undiluted, without the slightest sign of either local or general reaction. Following this injection she had no more chills and the highest the temperature reached was 101. The patient felt much better and clamored to go home. However, the temperature ranged from normal to 101 until the twenty-third of January, when I gave her a second injection of 10 c.c. of metaphen. Following this, the temperature promptly dropped to normal and on the 25th she was discharged.

At home the patient checked her temperature every four hours for the following three weeks, but the temperature in no instance went above 98.6. On February 18, I rechecked the blood to see what had happened to the agglutination titer and found it to be present in a dilution of 1:2560. This was checked in two different laboratories using three different strains, and in all instances the reports were similar. The patient has reported back to the office regularly for observation, but she has had no more subjective or objective symptoms of undulant fever.

### COMMENT

A case of undulant fever is presented which was observed from its onset, and which was treated with metaphen 1:1000 intravenously. The clinical duration of the disease was exactly ten days, and the temperature became normal six days after the use of metaphen was instituted. No other drugs were used. No reactions followed the use of the drug. The case is presented because of the apparently prompt response and to stimulate the further use and study of the drug in such cases. The fact that the drug was first used shortly after the clinical onset of the disease may account for the good response and one can only conjecture what results might be expected in a chronic case of undulant fever. However, its use in all cases seems indicated. The high agglutination titer one month following discharge from the hospital is striking.

\*From the Medical and Surgical Clinic, Fargo, North Dakota.

# CASE REPORT

## MANAGEMENT OF OPEN SAFETY PIN IN THE DIGESTIVE TRACT

GEORGE EARL, M.D.  
*Saint Paul*

Baby Elizabeth M., ten months of age, was first seen October 12, 1932, at about 10 a. m., with a history of severe cough, listlessness and fever. The mother thought that the child had swallowed a safety pin be-

present on the chest and back. This rash resembled one of scarlet fever but was not typical. The eyes were normal. The throat was reddened, and there was a nasal discharge. There was no vomiting. Her temperature was 101 degrees. The lungs were normal and the heart also except for a tachycardia. The abdomen was distended. There was no neck rigidity and neurological examination was negative, including a negative Kernig.

An x-ray plate (Fig. 1) revealed an open safety pin

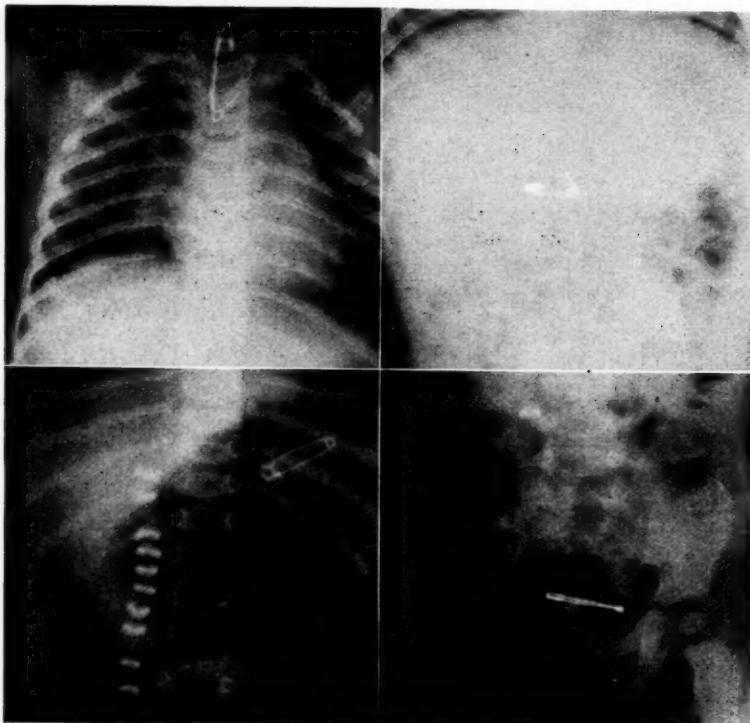


Fig. 1. (upper left). Pin in esophagus.  
Fig. 2 (upper right). Pin in stomach. Clasp in pylorus and point in stomach wall. Barium present in stomach.  
Fig. 3 (lower left). Pin closed in stomach. Second day post-operative.  
Fig. 4 (lower right). Pin in rectosigmoid. Fourth day post-operative.

cause there had been one missing from the bed sheet two days previously. However, she had not seen the child handling the pin. There was nothing else of importance in the history.

Physical examination showed a well-nourished child with a severe and constant cough. Restlessness and irritability were outstanding. An erythematous rash was

at the level of the first and second dorsal vertebrae with the blind end pointing downwards. Later fluoroscopic examination at 6 p. m. the same day showed the pin in the stomach, and, therefore, it was decided to treat the child conservatively in the hope that the pin would be passed. The child was taken home and apparently was quite well for three days.

Four days later (October 16, 1932) the patient was admitted to the hospital, not having passed the pin. The mother complained that the child had been refusing her feedings. Further examination was essentially negative except for a markedly distended abdomen. Laboratory findings were negative. X-ray examination (Fig. 2) with aid of barium by mouth showed an open safety pin at the outlet of the stomach, apparently straddling the pyloric sphincter, fixed and showing no tendency to move on gentle palpation. Tenderness over this area was apparently marked. Temperature, which had been approaching normal during the intervening days, had risen to 102.5 degrees.

With these findings, a laparotomy was performed under a combined local and gas anesthetic. The stomach was found to be distended with gas, which was released by means of small tube by mouth. The pin could then be palpated in the pylorus apparently straddling the sphincter. The pointed end was projecting downwards towards the pancreas, and the opposite end towards the gallbladder. The protecting sheath was in a direction corresponding with that of the pointed end. Cautiously the pin was manipulated back into the stomach, closed, and left there. Neither stomach nor bowel was opened, and there was no evidence of perforation or peritonitis.

The incision was closed in routine fashion without drainage. Immediate post-operative condition was very satisfactory and continued so. Immediate treatment consisted of sedatives (codein) and fluids (hypodermoclysis) to tolerance. There was no vomiting, and feedings were restored on the first post-operative day. An x-ray checkup was done on the second post-operative day (Fig. 3) which showed closed safety pin in the proximal end of the stomach. Barium given previously was then in the rectum, showing a normal motility. Temperature and pulse returned to normal late on the third post-operative day and continued so until discharge. Another x-ray (Fig. 4) was made on the fourth post-operative day which showed the pin in the rectosigmoid.

The pin was recovered on the fifth post-operative day following an enema, and the patient was dis-

charged on the sixth day with recovery apparently complete.

Dr. Greth Gardiner, consulting endoscopist, had suggested in the event of a subsequent laparotomy that the pin be closed through the bowel wall and left to pass, thus greatly decreasing the possibility of an operative death. Children of this age are known not to tolerate opening of the bowel, and while there is probably less risk in opening the stomach than the duodenum, the risk should be avoided if possible.

At the time of the operation, as far as Dr. Gardiner knew, the suggestion of closing the pin and allowing it to be passed was independent thinking. Dr. Gardiner had made an extensive search of the literature three years previous, and at that time such a procedure had not been suggested. His search of the literature had followed a peritonitis death from the removal of a safety pin in the bowel, and at that time the thought had come to him that it would have been better just to have closed the pin and let it pass in the natural way. This case, in which he was called in consultation while the pin was in the esophagus, offered the first opportunity to make the suggestion.

We recently note in the March 11 issue of the *Journal of the American Medical Association* a report of Dr. Harry Otten, of Springfield, Illinois, of a case operated October 4, twelve days previous to our case, in which Dr. Otten closed the safety pin in the stomach, fed it into a stomach tube, and removed the pin by that method. He states that he debated at the time whether to remove the safety pin through a gastrotomy or to let it pass the natural way, and finally decided on the method of using a stomach tube.

A newspaper report of April 6 from Chicago reports that Dr. Gustav Herpe, on April 4, closed a safety pin in the stomach of a child, which pin the child later regurgitated.

The purpose in making this report is to credit Dr. Gardiner's suggestion as one of independent thought previous to any report in the literature, and also to suggest that a closed safety pin of this size is so certain of natural passage that any manipulation beyond its closure is probably not indicated.

## PRESIDENT'S LETTER

AT this time the attention of the physicians of the northwest, and especially the members of the State Medical Association, should be directed to the eightieth annual meeting, which occurs at Rochester on May 21, 22, 23 and 24.

The meetings of the State Medical Association have, from year to year, been assuming a greater magnitude and have come to be the outstanding scientific and economic event in medical circles in this, and surrounding states.

The preliminary draft of the program for this year assures us that this will be, beyond doubt, the greatest meeting the association has ever held, and offers a splendid variety and choice of scientific presentations, which no medical man can possibly afford to miss.

This meeting at Rochester will be excellently housed, with ample and convenient space for all lectures, exhibits, demonstrations, and moving pictures.

A new departure, and one which should attract great interest, is the Monday program which is put on by eight of our statewide societies of specialists, the material to be presented in such a way, and of such nature as will be of great value to the general practitioner.

With the creative genius of our secretary, E. A. Meyerding, and the facilities of the Mayo Clinic at their disposal, the committee in charge has arranged a program of science, economics, entertainment, and sports, which will long be remembered by those who are fortunate enough to be able to attend.

Hoping to see all of you there, I am,

Yours very truly,



President,  
Minnesota State Medical Association.

# EDITORIAL

## MINNESOTA MEDICINE

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## THE STATE MEETING

Time marches on apace. The month of our annual State meeting has arrived. Never before in our recollection has the annual meeting been held in Rochester. What better spot for a get-together than this small Minnesota town so famous as a medical center?

Glance through the scientific program which appears elsewhere in this issue and, if in the array of addresses, demonstrations and scientific exhibits you can find nothing of interest, plan

to absent yourself. Even the movies will be in evidence in their new field of usefulness.

A five-ring circus will have nothing on our meeting this year with four and five societies of specialists performing simultaneously. "You've paid your money, take your choice."

There will be no banquet this year. Although to some this omission will be a source of real regret, to others there are better ways of spending the dinner hour on a nice summer's evening.

Medical economics will feature the Monday evening assembly. That entertaining medical editor, Dr. Morris Fishbein, ably assisted by the inimitable Dr. Charles H. Mayo, will expound to us on the "Trends of Medical Practice." Some think it is fast going to the dogs. We shall see. We are all interested in the subject of legislation and shall have the opportunity of obtaining first-hand information from the chairman of our Legislative Committee, Dr. Herman Johnson. Dr. A. W. Adson, president of the State Board of Medical Examiners, will also inform us of the recent activities of this body.

At the Tuesday evening meeting Dr. G. B. Eusterman will welcome us in behalf of the Olmsted-Houston-Fillmore County Society—hosts of the occasion. Mrs. A. A. Passer, president of the Women's Auxiliary, will report on the activities of this valuable organization. There is no one better posted on our State Association activities than our president, Dr. N. O. Pearce. While medical economics are closely linked with the deplorable economic state of affairs in the world at large, there are certain subjects in the lime-light at present of vital importance to the medical world. We assume that Dr. Pearce will touch upon these matters. Those who have heard Archbishop John Gregory Murray of Saint Paul know that here is a man who knows whereof he speaks. His message is sure to be inspiring. The outstanding visitor who will address the convention is Dr. Dean Lewis, president-elect of the American Medical Association. We shall all be interested to hear what is going on at the national association headquarters.

With Rochester so easily accessible and so lit-

tle need for us at home, a large and enthusiastic meeting is in the offing. Let's go!

### LOSS AND REPLACEMENT OF FLUID AFTER BURNS

In 1923 Underhill and his associates demonstrated the increase that may occur in concentration of blood of severely burned patients. They expressed the belief that this change was the result of loss of fluid through the burned area and that the fluid was plasma, or at least modified plasma. Recognition of these facts suggested to them a rational method of treatment for this condition, which consisted chiefly of administration of large amounts of fluid, some of which was in the form of physiologic saline solution. The results that they obtained were striking. Their work has been confirmed by subsequent extensive investigation, much of which has been done by Underhill and his co-workers.<sup>4</sup> It has been shown that the increased concentration of blood following a burn is the result of loss of fluid from the blood through increased permeability of capillaries in the burned area. Unexpectedly large quantities of fluid may be lost in this way.

In experiments on animals, superficial burns involving approximately one-sixth of the surface of the body have been inflicted under anesthesia, and have resulted in loss of water from the blood stream, amounting to as much as 70 per cent of the total volume of blood. Probably in more extensive burns the loss of water is even greater. Reabsorption of this fluid is exceedingly slow during the first few days after a burn, indicating that the increased permeability of the capillaries is effective in only one direction.

The fluid which is poured out on the surface of a burned area partakes of the nature of plasma. For this reason, decrease in the chlorides of the blood would be expected, and it does occur in many of the severe cases. The reason that it does not occur with more uniformity is because the organism possesses marked ability to compensate for losses of chloride. As much as 36 per cent of the sodium chloride of the blood may be lost without causing alteration in the chloride content of the blood.

Significantly increased concentration of blood

may result in failing circulation, oxygen starvation of the tissues, oliguria, and finally death. The observation has been made that concentration of the blood to 140 per cent of the normal value is, in a short time, incompatible with life, and concentration to 125 per cent of the normal value results in a precarious condition.<sup>3</sup>

Fortunately this condition is easily recognized by simple determinations of hemoglobin, and these should be made as a routine in all cases of serious burns. By prompt and appropriate measures to overcome the increased concentration of the blood, many patients recover who otherwise would die within the first few days as a result of this condition. Rational treatment consists in administration of adequate fluid, and salt, as suggested by the original observers,<sup>3</sup> and also in applications to the burns of such a substance as tannic acid, which markedly reduces the loss of water from the burned area.

The amount of fluid that is necessary varies considerably, depending on the severity of the case, but from 4,000 to 8,000 c.c. daily is usually advisable until the excessive concentration of the blood has been corrected. The larger amount designated is rarely necessary, now that the tannic acid treatment introduced by Davidson has become widely used.

Because loss of fluid through the burned area is constant, the intake of fluid should be as continuous as possible. Much of it may be given orally, but usually this is best supplemented by physiologic saline solutions given intravenously, by proctoclysis, or by hypodermoclysis. Large quantities may be administered intravenously by use of the continuous Murphy drip, allowing a period of from two and a half to three hours for administration of one liter. In most cases, if the foregoing plan of treatment is followed, improvement is soon noted; however, the concentration of hemoglobin should be determined frequently, and if the high concentration of blood and the accompanying oliguria persist, intravenous administration of acacia should be seriously considered.<sup>2</sup> In some cases, in spite of adequate administration of fluid, the fluid leaves the blood stream rapidly, and in such cases the acacia helps to hold the administered fluid in the circulation.<sup>5</sup> In all cases of shock or impending shock it is advisable to give acacia intravenously. It is best to give physiologic saline solution first, followed by

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acacia and then by more physiologic saline solution.

The occurrence of increased concentration of blood in many cases of severe burns is undeniable, and, in spite of all that has been written concerning it, is not generally appreciated. Probably the majority of patients with significant burns are still being treated without a single determination of hemoglobin being made and without it being recognized that concentration of the blood has increased. Fortunately, however, the practice of administering fluids freely in such cases has become general.

Although the tendency toward excessive concentration of the blood and lowering of the value for blood chlorides is present in all cases of extensive burn, actual changes in the blood are not demonstrable in all cases. There are several reasons for this. The burn may not have been sufficiently extensive, or prompt and adequate administration of fluids and local treatment to the burn to minimize loss of water may have averted the anticipated changes. Or the compensatory mechanism may have been adequate. It has been shown that the organism, without ingestion of food or water, possesses the ability to compensate for water lost to the area injured by a burn to the extent of at least 70 per cent of the blood volume.<sup>4</sup> But ultimately this mechanism breaks down, and when it does break increased concentration of the blood, with its dangerous sequelæ, occurs. The principles of treatment here outlined, therefore, should apply in all cases whether or not significant changes can be demonstrated in the blood.

E. G. B.

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#### THE RELATION OF TOBACCO TO CIRCULATORY DISTURBANCES OF THE LOWER EXTREMITIES

The association of excessive smoking, particularly of cigarettes, with thrombo-angiitis obliterans has been noted frequently, although the exact etiologic relationship to this fairly definite clinical and pathologic syndrome is still debatable. A statistical study made at The Mayo Clinic a few years ago in 350 cases of thrombo-angiitis obliterans revealed that only 1.4 per cent of the patients had never used tobacco whereas 61 per cent smoked cigarettes excessively, as compared with 26 per cent who did not smoke, and 33 per cent in a control group of 350 male subjects of similar ages who smoked excessively. However, when one considers that thrombo-angiitis obliterans is not a common disease, one is forced to the conclusion that its incidence among all heavy smokers of cigarettes is rare. Also, in spite of the great increase in cigarette smoking among women in recent years, the disease is still extremely rare among them.

Recently, some interesting work has been done by Sulzberger and by Harkavy, Hebdal and Silbert in determining the hypersensitivity of the skin of patients with this disease to tobacco extracts. Sulzberger found this hypersensitivity in 77 per cent of his series of cases of thrombo-angiitis obliterans and in 36 per cent of a control series. Harkavy, Hebdal and Silbert noted even more striking data: hypersensitivity of 89 per cent of patients compared with only 10 per cent of their control subjects. Nicotine seems to have been excluded as a factor. Hypersensitivity to tobacco could possibly explain many of the riddles in this disease, but one must be careful not to jump to this explanation as there is a gap between skin allergy and the rather distinctive pathologic changes found in the blood vessels. Also, there remains a definite group of patients with thrombo-angiitis obliterans who do not seem to be skin-sensitive to tobacco. Further studies of larger and more varied groups of cases are desirable.

Another report published within the last year by Maddock and Coller may be even more significant. They found that twenty subjects, both light and heavy smokers, had a definite drop in cutaneous temperature of the digits following the smoking of three cigarettes during a standardized

test. After filtering the smoke and using other types of smoke the effect was not noted and it was concluded that absorbed portions of the tobacco smoke were responsible. The fall in cutaneous temperature can only be interpreted as being due to vasoconstriction. If this work is verified it would seem that even if tobacco smoking should not be proved to be the direct cause of thrombo-angiitis obliterans, it is certainly an unfavorable influence and in a case with markedly limited blood supply or with an acute exacerbation, it might turn the scales in favor of gangrene or other trophic lesions. Tobacco also might be deleterious in other peripheral vascular disturbances both vasospastic and occlusive. Furthermore, if the vasoconstriction is due to sympathetic stimulation, as would seem quite possible in view of some other known effects of tobacco and nicotine, one may consider the influence of smoking on other clinical syndromes associated with an overactive or irritable sympathetic nervous system.

Purely clinical observation has tended to show that some patients with thrombo-angiitis obliterans seem to improve and have a more benign course if they stop smoking. There are too many exceptions to incriminate tobacco smoking as the sole etiologic agent, but, considering present knowledge, it seems reasonable to indict it as at least an unfavorable influence. One should strongly insist, although it is often futile, that patients suffering from thrombo-angiitis obliterans discontinue using tobacco completely and permanently.

NELSON W. BARKER, M.D.

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#### FEVER THERAPY IN NEUROSYPHILIS

The suggestion of Wagner von Jauregg, made more than ten years ago, that inoculation with *Plasmodium vivax* would offer results in the treatment of general paresis that had not been accomplished by other methods of treatment was a revolutionary idea in antisyphilitic therapy. The results which he reported in his first publication have been substantiated in a large series of cases by observation now for almost a decade. However, the successful results obtained with this treatment of a heretofore extremely resistant complication of syphilis are by no means the outstanding results of Wagner von Jauregg's suggestion, for it has been shown that malaria ther-

apy is not only the most effective means available of treating general paresis but that it is even more efficient in preventing the development of general paresis in cases of neurosyphilis. Accordingly, patients who have undergone intensive treatment for neurosyphilis with the arsenicals, mercury, and bismuth, and who have failed to show a satisfactory serologic response, are ideal candidates for treatment with malaria therapy.

Experience with malaria therapy also has called to the attention of syphilologists the fact that the individual's "resistance mechanism" to syphilis, a factor which was lost sight of in the enthusiasm attendant on the introduction of arsphenamine, is of decided importance in the outcome of neurosyphilis. Hence, renewed efforts to stimulate this defensive mechanism have been the outgrowth of the treatment with malaria therapy. A variety of fever-producing agents, including vaccines and electrical fever-producing machines, have been developed. Electric appliances such as super-diathermy and radiotherapy machines or electric blankets offer the advantage that patients can be treated by physicians who see only an occasional patient with neurosyphilis, that the degree, duration, and frequency with which the fever is produced may be more easily controlled, and that the treatment may be given almost at will. These advantages apply to all forms of electric fever-producing methods. Neither method, however, is without risk, since in cases in which the central nervous system is involved the deleterious effects on the heat-controlling centers of the nervous system are no more readily prevented when these electric measures are employed, than they are when malaria therapy is used. It is difficult to administer the electric treatments to the patient whose disease is not sufficiently advanced for the mental disturbances of general paresis to have been exhibited. The demented patients can be readily treated by forceful measures, whereas the patient who has not yet reached the stage of general paresis, as a rule objects rather strenuously to the ordeal of having his temperature raised by electricity. There is an additional factor which further observation may or may not support, and that is, remissions from fever therapy are less frequent and not quite as complete following the electric method of producing fever as they are from malaria therapy. In justice to these electric methods, however, a decade must elapse before concrete opinions will be available as to their

value. Just as with malaria therapy, time and observation of the results of treatment in a large series of cases are essential in determining the good as well as the bad effects of these electric fever-producing agents.

PAUL A. O'LEARY, M.D.

#### CORRECTION

On page 227 in the April issue of MINNESOTA MEDICINE in an article on "Carcinoma of the Esophagus" by Dr. E. C. Bartels, the word "without" was used in describing Jackson's method of dilatation of the esophagus. The sentence should have read: "The blind method of dilatation as used by Jackson with the esophagoscope is hazardous and should be avoided."

## OF GENERAL INTEREST

Dr. H. T. Sherman, formerly of Grand Meadow, Minnesota, is now located in Bellingham, Minnesota.

Dr. Leo M. Crafts, Minneapolis, has recently been notified of his appointment as a member of the Advisory Council of *The Living Age*, which has just been established by the Editorial Board of that periodical.

Harold E. Hullsiek, St. Paul, addressed a joint meeting of the Redwood-Brown County Medical Society and the Union Hospital Staff of New Ulm at the hospital, April 4, on the subject of Rectal Diseases.

Dr. Henry E. Michelson, Minneapolis, delivered the address before the annual meeting of the Wayne County Medical Society, in Detroit, Michigan, on April 18, 1933. The subject of his discussion was "Cancer of the Skin."

A special bus fare of \$2.35 for the round trip is offered by the Jefferson Transportation Company to physicians and others from the Twin Cities attending the State Society meeting at Rochester, May 22, 23 and 24. This rate is offered only if a special bus is reserved seating thirty-three persons and filled to capacity.

At a recent meeting of the New York State Medical Society at the Waldorf Astoria Hotel in New York City, Dr. H. Hamilton Cooke of Lowville, New York, was awarded the annual medal and prize for his thesis on "A Pathological, Experimental and Clinical Consideration of Lipoid Deposits in the Gallbladder."

Dr. Cooke formerly lived in Hutchinson, Minnesota, and was a Fellow in Surgery at the Mayo Clinic, Rochester.

Dr. Charles R. Drake of Minneapolis is among the list of those in line for nomination, May 8, as a member of the Board of Education of Minneapolis. As a physician Dr. Drake is particularly interested in public health measures as well as education in general. The interest of physicians in public activities of this sort is commendable.

Donald A. Laird, director of the psychological laboratory of Colgate University, has been appointed to supervise the exhibit on sleep and fatigue for the Century of Progress Exposition, opening at Chicago in June. The exhibit, which is being supported by the Master Bedding Makers of America, will feature the actual use of apparatus used on recent studies of sleep and fatigue at several universities, giving visitors an opportunity to take some of the tests. It is expected to accumulate in this way a mass of data bearing on the diurnal course of fatigue.

The following amendments to the constitution of the Minnesota State Medical Association were passed by the House of Delegates of that body at the annual meeting in St. Paul in May, 1932. Under a provision of the constitution these amendments will go into effect one year after their passage, following the 1933 meeting of the State Association.

*Article IV, Section 4:* Affiliate members should be those members of component districts or county medical societies, who upon their own request, and having held membership for a period of twenty-five years in this Association, and having reached the age of seventy years, or who through physical disability are unable to engage in active practice, and who shall have been declared affiliate members of their own district or county medical society at its regular meeting, such action having been approved by the Council.

*Article V: House of Delegates:* The House of Delegates shall be the legislative and business body of the Association and shall consist of (1) Delegates elected by the component county and district societies, (2) Councilors, (3) The President, (4) The President-Elect, (5) Ex-officio Secretary and Treasurer, (6) Past Presidents who shall be entitled to the privileges of the floor but without the right to vote.

*Article VI: Council:*—The Council shall consist of the Councilors, the President, the President-Elect, the immediate Past President, and ex-officio the Secretary and the Treasurer. Besides its duties mentioned in the by-laws, it shall constitute the Finance Committee of the House of Delegates. A majority of Councilors shall constitute a quorum.

#### MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

##### EX-NATUROPATH AND PHYSICAL CULTURIST SENTENCED TO WORKHOUSE

State of Minnesota vs. Mitchell Judy  
On March 29, 1933, Judge Levi M. Hall, of the Hennepin County District Court, sentenced Mitchell Judy, twenty-eight years of age, to serve six months in the Minneapolis workhouse. Judge Hall refused to suspend the sentence because of the fact that Judy has a previous conviction for a similar offense committed in Minneapolis in 1932.

Jurdy was tried before a jury which deliberated only forty-five minutes in bringing in its verdict of guilty. At the time of his arrest Judy was located at the Rosedale Hospital, 4429 Nicollet Ave., Minneapolis. The complaint against Judy charged him with prescribing and suggesting a form of treatment for Mrs. Katherine Auge, 2835 Central Ave. N. E., Minneapolis. Mrs. Auge paid Judy and the Rosedale Hospital a total sum of \$158.00. Mrs. Auge has been suffering for sometime from Hodgkin's disease. Dr. C. C. Carpenter, a licensed physician, testified on behalf of the defendant, that he was the physician in charge of the Rosedale Hospital, and that Mrs. Auge was his (Dr. Carpenter's) patient. This was vehemently denied by Mrs. Auge. The testimony disclosed that Mrs. Auge was placed upon an orange juice diet and given light ray treatments and some massage. The treatments lasted a period of fourteen days.

On February 19, 1932, Judy pleaded guilty to a similar offense and received a suspended sentence of one year in the Minneapolis workhouse. While he was on probation the present case occurred. Judy has professed to be a naturopathic physician and physical culturist.

The investigation of this case was handled by the Minnesota State Board of Medical Examiners in cooperation with the Better Business Bureau of Minneapolis. Judge Levi M. Hall, before whom the trial was had, presided over the same in a very fair manner. The defendant was accorded his rights and at the same time a fair instruction was given to the jury by Judge Hall in reference to the law on this subject.

## A FORUM OF THE COMMITTEE ON PUBLIC HEALTH EDUCATION

### Costs of Medical Insurance

One of the unfortunate by-products of the widely published majority report of the Committee on the Costs of Medical Care has been the emergence of a large number of commercial schemes for health insurance.

This development was foreseen at the time of the publication of that report and the public was duly warned. Physicians, having been put in the light of selfishly interested parties, cannot expect that their warnings will be universally heeded.

So far, the lay promoters have been halted to some extent by the ruling of the attorney general's office that health insurance is subject to the same laws that regulate life insurance.

Early in April, however, a new company calling itself the Pioneer Mutual Health and Benefit Insurance Company opened "home offices" in the New York Building in Saint Paul with R. H. G. Whatley, president; Sidney O. Lankester, vice president, and Samuel Neumann, secretary and treasurer.

The plan under which this new company is to operate, as quoted in the *Saint Paul Dispatch*, of Monday, April 10, is as follows:

"Policies will be sold to heads of families covering all dependent members of the family living under one roof.

"The services provided include medical treatment for all sickness or accidents to be given in the office of the attending physician or hospital. Necessary operations will be performed under the policies as well as necessary dentistry, the latter listed as non-gold fillings work, extractions and cleaning."

Mr. Neumann, secretary and treasurer of the company, is quoted to the effect that "medical and dental services will be given by a staff of doctors and dentists regularly maintained by the company" who will doubtless be the judges of what are and what are not "necessary operations."

Presumably this company is organized and capitalized to conform with state and federal insurance regulations. Presumably, also, its backers believe that the hiring of a staff of doctors in these hard times will be the least of its difficulties. The chances are, however, that no matter how well organized the company may be, it will be extremely difficult to hire reputable physicians.

Physicians rightly look with suspicion on any commercial scheme for providing medical care. In such cases, funds which should be spent exclusively on medical care must be made to yield profits to lay promoters. As the Bureau of Economics of the American Medical Association has repeatedly pointed out, approximately 40 per cent of the money paid by subscribers for all such medical insurance schemes goes, not for medical and hospital care at all, but to cover overhead of organization, salaries of salesmen, profits on investment.

The physician can see no adequate reason for making such costly experiments when better care at lower costs can be had directly from a well qualified physician. He must reckon, however, upon an unusually susceptible public and upon a continuous newspaper propaganda favoring reorganization of medical practice which nicely paves the way for commercial sorties into the medical field.

Certainly it should be the responsibility of the organized medical profession to protect the public against questionable schemes of medical insurance.

Four outstanding speakers will discuss the situation of the doctor and of medical practice at the 80th Annual Meeting of the State Society at Rochester on the evenings of Monday and Tuesday, May 22 and 23. They are Morris Fishbein of Chicago, Archbishop John Gregory Murray of Saint Paul, Dean Lewis of Baltimore, president of the American Medical Association, and N. O. Pearce of Minneapolis, president of the State Society. These discussions, as well as the scientific sessions of the 22nd, 23rd, and 24th (for details see the program printed elsewhere in this issue), should be of great importance to every man who is practising medicine.

## MISCELLANEOUS

### AN AVOCATION FOR THE MALE CARDIAC INVALID

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This note is on therapy, psychotherapy perhaps, for application to the severely ill, but still ambulatory, adult, male, cardiac invalid who before his illness was an ardent follower of competitive sport. Although I have had the suggestion in mind for several years, ever since a pathologist who is an expert rifleman introduced me to a highly technical and satisfying hobby, I have not presented it. However, I have now been urged to do so by a well known cardiologist.

The patient with an injured heart, who has gone through the period of rest and other treatment recently recommended by Kilgore,\* in time is likely to be allowed to walk about. Then, if he has been a sportsman, he will see little to live for. Golf, fishing of any interest, game fishing, even trap-shooting are over for him. All are too fatiguing. Bridge and checkers will pall on a formerly physically active man. Mental depression tends to descend on him, and he will be likely to give up his life in a burst of rebellion against the regimen, unless he can be provided with a manly, competitive, out-of-door sport.

An article by Roberts,† although it was not written with the invalid in mind, contains the solution. It is rifle shooting from a bench rest. Shooting in the four standard positions, standing, kneeling, sitting, and prone, would not do for the patient with heart disease although it might do for persons with less serious chronic illnesses. These positions are fatiguing. However, in shooting from a bench rest the patient sits on a chair, before a table built with an "L" shaped jog in one end (Fig. 1). His arms rest on the table, his thorax is supported by the limbs of the "L" shaped jog, the barrel of the rifle is supported by the muzzle rest. The only way the patient conceivably can injure himself is by holding his breath too long before squeezing the trigger. He can guard against this remote hazard and will be inclined to do so if he can be kept cheerful.

In shooting a rifle in this way, the patient who now is a rifleman and again a sportsman, converts himself into a part of what riflemen call a machine rest, in so far as he can. The fact that he is not a machine, however, is where the enjoyment comes in. The modern 22 caliber target rifle, which is as large an arm as the patient should use, is a scientifically accurate instrument that will bring joy to any sportsman's heart. To him it is a beautiful object and the physician can be assured that it has practically no recoil. The ammunition that has been developed for this rifle is both accurate and cheap. However, with all these refinements, both rifles and ammunition are made by men and are used by men under varying conditions of range, light, and wind velocity. Let the rifleman spend a fortune on micrometer sights, telescopes, special barrels, stocks, actions, and loads, whatever he does his score will never become uniformly perfect. He will shoot differently from day to day, for no discernible reason, and if he should get too expert to suit himself, he need only change his rifle, his range, his sights,

or his ammunition to find a new set of problems facing him.

In fact, no man can master, in a lifetime, all there is to know about rifles and rifle shooting. There is reading, study and light bench work enough to occupy the rainy days, and not any man who masters even a lit-



Fig. 1. Shooting from a bench rest. (Courtesy The American Rifleman.)

tle of the knowledge and technic that centers about the rifle need ever be lonely. There is a national organization of riflemen, and riflemen in any community soon become acquainted. The talk among them is endless, some of it very technical indeed, and conducive to anything but introspection.

The physician need not feel hesitant about presenting these views, even to a former sportsman, for many out-of-door men are not acquainted with rest shooting nor with new developments in small caliber rifles and ammunition.

### HOSPITALETE\*

GEORGE B. LAKE, M.D.  
Chicago, Illinois

There are three entirely ethical ways in which a physician can make himself and his work known to his confreres and to the public: He can develop his skill and knowledge in some one or several lines to the point where his success as a healer will be so outstanding that his fame will spread from mouth to ear; he can prepare himself to talk so interestingly and instructively on one or several subjects that his services as a speaker, in person or over the air, will be eagerly sought; or he can embody his researches, experiences or cogitations in written articles, for publication in the professional or lay press. For most men, the last-named method is the easiest, but even that is no God-given endowment. It requires real and sincere study and work.

While it is quite obvious (especially to foreigners who are trying to learn it) that the English language contains many inconsistencies of spelling, pronunciation and construction, the fact remains that, when it is thoroughly known and used with intelligence and discrimination, it is one of the most flexible and elegant methods for conveying ideas from one man to another.

Physicians are supposed to be—and very generally

\*Kilgore, E. S.: Treatment of acute coronary occlusion. *Jour. Am. Med. Assn.*, 100:315 (Feb. 4), 1933.

†Roberts, N. H.: Rest shooting. *American Rifleman*, 81:5 (Feb.), 1933. Submitted for publication April 15, 1933.

\*Reprinted by permission from the *Medical Mentor*, January, 1932.

are—educated and cultured men, and their spoken communications, as a rule, substantiate this supposition; but when nine out of ten medical men sit down to write a professional article, they appear to forget that they have ever had any training or practice in grammatical construction and lapse into a dialect that is in the class with the argot of the yeggs and hijackers.

In writing up a medical history, in the wards of a hospital or in the office, the busy intern or practitioner finds it convenient to employ certain symbols and abbreviations which are fully understood by himself and by the other physicians and nurses who may have occasion to consult these records. Such notations are not supposed to be English, but merely a form of time-saving shorthand, which serves its purpose excellently.

This is all well and good—until some ambitious man, with a proper and highly laudable desire to see his name and the results of his labors in print, attempts to embody the data from these case records in an article, and feels that he has done so when he has made verbatim copies of the data on the charts. Then the stuff is a mess.

Some misguided would-be authors seem to feel (obviously they do not *think*) that the way to write a technical paper is to leave out all of the articles, prepositions and conjunctions and most of the adjectives, as well as the predicates of about half of the sentences, and to use as many abbreviations as possible. B.M.R., C.A.C., t.i.d., T.A.T., R.O.A., and similar cabalistic signs will do for history sheets, but not for the pages of a high-class medical journal, unless the words they represent are to be used frequently in the same paper, and they should be written out in full the first time and followed by the abbreviation in parentheses, after which the latter may be used alone.

We have all heard wise and able men, who should have (and no doubt did) known better, in the course of a line of shop-talk in the scrub-up room, make observations like this: "He had no temperature and his Wassermann was negative, so I operated the case and removed the pathology." This is not English: It is hospitalese.

When such a melange of shocking barbarisms is emitted in the course of more or less intimate dialogue, there may possibly be some *excuse* (though there is never any sound *reason*) for it, even though it corrupts the speech, and with it the thinking, of younger men who may hear it. But when such examples of mangled syntax are permitted to appear in formal addresses or articles, there is neither reason, excuse nor any possible justification for such a lazy, sloppy and wholly reprehensible practice.

Everything not at absolute zero (minus 273° Centigrade) has *temperature*, so every human being has a good deal of it. A sick man may have an *elevation* of temperature above the normal, which is simply and properly called *fever*.

There is no such object in nature nor concept in philosophy as a *wassermann*. There was once a *Doctor Wassermann*, whose test for syphilis is widely used and whose name is properly applied, as an *adjective*, in describing the *test*.

One may *operate* a jig-saw or a peanut roaster, but not a man. A surgeon can *operate upon* a patient, not upon a *case*, which latter word is merely an abstract symbol denoting a particular instance or example of some disease or morbid process.

*Pathology* is the science or study of the nature and results of disease—an intellectual concept; not a material object which can be seized with forceps and ablated with a scalpel. It is ridiculous to say "The lungs showed pathology," as it would be to remark that the throat contained laryngology or the bladder urology. The organs may show *pathologic changes or lesions*.

Following up this last line of thought, it is an

equally loose locution to speak of the body as containing or exhibiting *chemistry* (another science). Blood chemistry, for example, is an abstract idea and cannot "show" anything; though blood-chemistry *studies* can, and often do, give us valuable information. Serology is still another science, and can no more be "positive" or "negative" than can arithmetic. Those terms are properly applied to serologic *findings*.

Launching a general attack upon a few of the most popular and pernicious errors of form and construction which one hears in medical talks and sees in professional journals, "tubercular" means, characterized by the presence of nodules (we may have tubercular leprosy). An individual infected with the tubercle bacillus is *tuberculous*.

The words cystoscope, urethroscope, laryngoscope and such are *nouns*, symbolizing certain surgical instruments, and to use them as verbs is a shameful exhibition of professional provincialism. It is as ludicrous to say that a patient was "cystoscoped" (or "x-rayed" for that matter) as it would be to declare that he was "speculumed" or "scalped."

The appendix, the abdomen and the frontal sinus are definite, objective parts of the human body. They may be large or small, normal or abnormal; but they can never be "acute" or "chronic," as these latter terms refer to *processes or diseases*, not to organs. One is liable to suffer from acute *appendicitis*—never from an acute appendix (or abdomen or frontal sinus).

Diabetic, prostatic and neurotic are *adjectives*, like ecstatic and parabolic, and it is very doubtful if there is ever any justification for using them as substantive nouns. It is far better practice to speak of a diabetic, prostatic or neurotic *patient*. "Diabetic surgery"—meaning operations upon diabetic patients—is entirely beyond the pale, being not at all analogous to "prosthetic surgery" which, properly used means surgery upon the *prostate gland*.

Intravenous, intramuscular and hypodermic are *adjectives*, properly applied to various types of parenteral injections, and should never be used to describe the drugs so administered (as "intravenous iron") nor as substantive nouns. There is no more justification for saying "a hypodermic of morphine" than there would be for "an intravenous of arsphenamine." For a physician to speak of any type of parenteral injection as "a shot," in any except the most utterly informal circumstances, should be enough to insure the revocation of his license to practice.

Many more examples of medical solecisms, provincialisms, lazysisms and other types of professional jargon and hospitalese could be adduced, but these should be sufficient to set any perspicacious man thinking and turn him from his evil ways (if any) to the dictionary, the thesaurus and the textbooks of grammar and rhetoric.

Heywood Broun is quoted as having said that the essence is more important than the form. If that is true, a correspondent of the *Chicago Tribune* remarks, then Mr. Broun should not find fault if a man says, "If I'd a knowed I coulda rode I'd a went. But if I'd a went I couldn't of et."

That may seem like an exaggerated instance, but considering the difference, in educational and environmental background, between the perpetrator of this horrible example of cruelty to the English language and the average physician, the things that editors find in certain manuscripts (some of which even leak through into the printed pages) are just as bad.

The man who aspires to achieve any degree of fame as a medical writer should familiarize himself, not only with the principles of English grammar and composition, but also with the usages employed by the best writers in this field, and should avoid, as he would a pestilence, any tendency to lapse into hospitalese.

—*Medical Mentor*, January, 1932.

Dr. J. physician from a Holland practice a heart Rite M. Ramsey Medical Surgery fraterni son, H. Mrs. A. Margar

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## OBITUARY

### John Archibald Cameron

1869-1933

Dr. John Archibald Cameron, prominent St. Paul physician, died Tuesday, April 4, 1933, in his home, from a heart attack. He was the husband of Jane Holland Cameron, noted singer. Dr. Cameron, who had practiced medicine in St. Paul since 1902, had suffered a heart complaint for about six months. A Scottish Rite Mason, Dr. Cameron also was a member of the Ramsey County Medical Society, Minnesota State Medical Association, and the Minnesota College of Surgery. He was a member of Alpha Kappa Kappa fraternity. Besides Mrs. Cameron he is survived by a son, Holland, a daughter, Phyllis Jane, and two sisters, Mrs. Anna Wheeler of Devils Lake, N. D., and Mrs. Margaret Nieland of California.

### Godfrey Deziel

1861-1933

Dr. Godfrey Deziel, aged seventy-one, practicing physician in Minneapolis for forty-five years, died Monday, March 27, 1933, at St. Andrew's Hospital. Dr. Deziel was born in Ontonagon, Michigan. He was a member of the Minneapolis Athletic Club and the St. Anthony Commercial Club, and was physician for Aerie No. 1247, Fraternal Order of Eagles. Dr. Deziel is survived by his wife, a daughter, Mrs. Stephen Darling of Appleton, Wisconsin; a grandson, Stephen D. Darling of Appleton; a brother, Henry Deziel, and a sister, Mrs. Ambrose Jubert.

### Clarissa Clay Richardson

Dr. Clarissa Clay Richardson, herself a physician and the wife of Dr. Harold E. Richardson, St. Paul, died Sunday, April 16, 1933. Besides her husband, she is survived by a daughter, Ann Marie, and an infant son.

Mrs. Richardson was a former resident of Minneapolis. She obtained her medical degree at Creighton University in Omaha in 1917 and served her internship in St. Mary's Hospital, Duluth.

She had been an assistant city physician in Minneapolis, serving at General Hospital, and a resident physician at the State School for Feeble Minded in Faribault. She was married in 1919 and ceased practice in 1920.

### WILLIAM PEPPER

William Pepper began life under conditions which are very often unfavorable to success. His father, a distinguished physician, the Professor of Medicine in the school in which his son was educated, belonged to a family of position and influence. For the young man there were none of those tempering "blows of circumstance," no evil star with which to grapple and grow strong. Quite as much "grit" and a much harder climb are needed to reach distinction from the top as from the bottom of the social scale, and to rise superior to the *res abundans domi* has taxed to the uttermost many young men in this country. We have heard enough of the self-made men, who are always on top; it is time now to encourage in America the young fellow who is unhappily born "with a silver spoon in his mouth." Like the young men in the Gospels, he is too apt to turn away sorrowfully from the battle of life, and to fritter away his energies in Europe, or to go to the devil in a very ungentlemanly manner, or to become the victim of neurasthenia. To such the career I am about to sketch should prove a stimulus and an encouragement.—Counsels and Ideals from the Writings of William Osler.

## REPORTS AND ANNOUNCEMENTS OF SOCIETIES

### MEDICAL BROADCAST FOR THE MONTH

The Minnesota State Medical Association Morning Health Service

The Minnesota State Medical Association broadcasts weekly at 11:15 o'clock every Wednesday morning over Station WCCO, Minneapolis and Saint Paul (810 kilocycles or 370.2 meters).

*Speaker:* William A. O'Brien, M.D., Associate Professor of Pathology and Preventive Medicine, Medical School, University of Minnesota.

The program for the month of May will be as follows:

May 3—Child Health—A Challenge.

May 10—Galvanic Mouth Burns.

May 17—Medicine of the Aborigines.

May 24—Dementia Praecox.

May 31—Periodic Health Examination of Women.

### RED RIVER VALLEY SOCIETY

Ulcers of the Stomach and Duodenum was the subject for discussion at the spring meeting of the Red River Valley Medical Society held at the Hotel Crookston, April 11, 1933. Dinner for members of the society and for the Women's Auxiliary preceded the meeting. The following doctors participated in the scientific discussion: W. F. Mercil, Crookston; O. E. Locken, Crookston; J. F. Normal, Crookston, and Edward Bratrud, Thief River Falls.

### RICE COUNTY SOCIETY

The Rice County Medical Society met Thursday, April 20, 1933, in Northfield. R. E. Scammon, Dean of Medical Sciences at the University of Minnesota, talked on The Relation of the University Hospital to the Medical Profession. Mr. J. M. Sweitzer of the Employers' Mutual Liability Insurance Co. talked on Workmen's Compensation as it Affects Medical Practice. Mr. F. M. Brist, attorney for the State Board of Medical Examiners, spoke on Recent Activities of the Board of Medical Examiners. Mr. Brist also reviewed the work of the Legislative Committee of the State Association.

### WABASHA AND WINONA COUNTY SOCIETIES

There were twenty-six in attendance at the annual dinner and joint meeting of the Wabasha and Winona County Medical Societies, held at Buena Vista Sanatorium, Wabasha, on Monday evening, April 10, 1933.

Dr. R. E. Scammon talked on "Your University Medical School and Hospital," and "Guild Medicine," Dr. N. O. Pearce, president of the State Medical Association, spoke on "Some Problems of Our Profession," and Mr. F. Manley Brist discussed the activities of the State Board of Medical Examiners and the work done by the Committee on Legislation of the State Medical Association at the Capitol this winter.

Lay members of the Bi-County Tuberculosis Commission were included among the invited guests.

### PROFESSIONAL SENSITIVENESS

More perhaps than any other professional man, the doctor has a curious—shall I say morbid?—sensitivity to (what he regards) personal error. In a way this is right; but it is too often accompanied by a cocksureness of opinion which, if encouraged, leads him to so lively a conceit that the mere suggestion of mistake under any circumstances is regarded as a reflection on his honor, a reflection equally resented whether of lay or professional origin.—Counsels and Ideals from the Writings of William Osler.

**MINNESOTA STATE MEDICAL ASSOCIATION**  
**EIGHTIETH ANNUAL SESSION . . . MAY 21-24, 1933**  
**ROCHESTER, MINNESOTA**

**Business Program**

**Sunday Afternoon, May 21**

12:30—Council.....	University Club
2:00—Reference Committee.....	University Club
7:00—House of Delegates.....	Sun Room
11:00—Council.....	University Club

**Tuesday Morning, May 23**

12:00—House of Delegates.....	Kahler Hotel (Luncheon)
1:45—Council.....	University Club

**Wednesday Morning, May 24**

General Assembly	
Nurses' Home, St. Mary's Hospital	
10:00—Presentation of Officers.....	Auditorium
Reference Committee	
F. H. MAGNEY.....	Duluth
W. F. BRAASCH.....	Rochester
J. C. HULTKRANS.....	St. Paul
C. A. MCKINLAY.....	Minneapolis
C. L. SCOFIELD.....	Benson

**Scientific Program**

**Monday Morning, May 22, 1933**

**TRUDEAU SOCIETY**

Room G 14, St. Mary's Hospital—9:00 A. M.

*Presiding Officers:*

President: A. T. Laird	Secretary: D. R. Hastings
Pathology of Tuberculosis—H. E. Robertson, Rochester	
Childhood Tuberculosis—J. A. Myers, Minneapolis	
Pulmonary Tuberculosis: General Treatment—E. S. Mariette, Oak Terrace	
Pulmonary Tuberculosis: Surgical Treatment—S. W. Harrington, Rochester	
Control of Tuberculosis in Minnesota—A. J. Chesley, Minneapolis	
Control of Tuberculosis: National and International—John H. Peck, President, National Tuberculosis Association, Des Moines	

**HEART ASSOCIATION**

East Hall, Nurses' Home—9:00 A. M.

*Presiding Officer:*

President: Frank J. Hirschboeck

Life History of a Patient with Mitral Stenosis.
Complications of the Final Stage: Autopsy Findings—(Lantern)—C. N. Hensel, St. Paul
The Treatment of Some Common Heart Disorders—A. R. Barnes, Rochester
Paroxysmal Tachycardia and Associated States—E. L. Tuohy, Duluth
The Production of the Electrocardiogram—(Cinema)—F. A. Willius and A. R. Barnes, Rochester

The Influence of Gravity on the Circulation—Frederick Scott, Minneapolis

Case Reports—T. A. Peppard, Minneapolis

Adiposity of the Heart—F. A. Willius and H. L. Smith (by invitation), Rochester

**DEPARTMENT OF OBSTETRICS  
AND GYNECOLOGY**

University of Minnesota

Room G-15, St. Mary's Hospital—9:00 A. M.  
Occiput Posterior: Lantern Slide Demonstration and Lecture—G. E. Hudson, Minneapolis

Version with Manikin Demonstration—L. W. Barry, St. Paul

Myomata of the Uterus—J. A. Urner, Minneapolis

Modern Idea of the Etiology and Treatment of Abortions—J. C. Litzenberg, Minneapolis

**MINNESOTA SURGICAL SOCIETY**

Auditorium, Nurses' Home—9:00 A. M.

*Presiding Officers:*

President: T. J. Kinsella Secretary: V. P. Hauser  
The Treatment of Burns—James Morrow, Austin

Chronic Intussusception of the Gastro-Intestinal Tract: Report of 39 Cases—C. W. Mayo, Rochester  
Retrograde Intussusception of the Jejunum Through a Gastro-Enterostomy Stoma—T. J. Kinsella, V. K. Funk, and Charlotte C. Van Winkle, Oak Terrace

Carcinoma of the Rectum—C. F. Dixon, Rochester  
Results of the Treatment of Bladder Tumors—V. S. Counsellor, Rochester

Therapy of the Artificial Menopause—R. J. Moe, Duluth

Presentation of Three Cases—Lipoma of Joint Capsule with Successful Removal—R. K. Ghormley, Rochester

Sterility and Fertility in the Menstrual Cycle (with demonstration of a new slide rule)—J. M. Culligan, St. Paul

Restoration and Conservation of Renal Function Following the Removal of Obstructing Lesions—Waltman Walters, Rochester

**TABLE DEMONSTRATIONS**

Nurses' Home—11:00 A. M.

East Hall

Practical Points in the Treatment of Rectal Disorders—L. A. Buie, Rochester

Animated Drawings of the Origin and Surgical Management of Anal Fistula

Auditorium Stage

Physiotherapy Demonstration—Arthritis and Acute Injuries—A. U. Desjardins, P. S. Hench, and Ann Kelley, R.N., Rochester

## West Hall—B-24

Complete Medical and Laboratory Examination of a Patient—W. C. Alvarez, Rochester

## West Hall—B-22

Exhibit on Radiotherapy, Roentgen Rays and Radium—A. U. Desjardins and H. H. Bowing, Rochester  
Presented by Section of Therapeutic Radiology, Mayo Clinic

## West Hall—B-20

Treatment and Complications of Gonorrhea—A. L. Clark, Rochester

Demonstrating the diagnostic technic, including staining of slides and irrigation of patient

## West Hall—Booth No. 6

Minnesota General Hospital—Relationship to the Medical Profession

## West Hall—B-27

Transurethral Punch Operation—H. C. Bumpus and G. J. Thompson, Rochester

## West Hall—Booth No. 24

Treatment of Acute Burns—R. K. Ghormley and E. G. Bannick, Rochester

Demonstration and Exhibit showing enlarged photographs, charts and equipment

## West Hall—Booths No. 21 and 22

The Occurrence of Tularemia in Man and Animals—R. G. Green, University of Minnesota

## West Hall—Booths No. 7 and 8

Professional Pharmacy as Applied to the Physicians—Under the auspices of the Inter-Professional Relationships Committee of Hennepin County.

## West Hall—B-26

Differential Characteristics of Malignant Cells—W. C. MacCarty, Rochester

## West Hall—B-28

Treatment of Syphilis—P. A. O'Leary and L. A. Brunsting, Rochester

## West Hall—Booth No. 27

(1) The Geiger Counter and Its Application to the Measurement of the Circulation Time of the blood; (2) The Electromotive Thermometer and the Measurement of Skin Temperature; (3) Short Electric Wave (Radiotherapy) Apparatus—Charles Sheard, Ph.D., Rochester

(This apparatus will be demonstrated to those who make application at the Registration Desk before 9 o'clock Monday and Tuesday)

**Monday Afternoon, May 22, 1933****NORTHWESTERN PEDIATRIC SOCIETY**

Auditorium, Nurses' Home—1:30 P. M.

President: Alexander Stewart Secretary: E. F. Robb  
Primary Carcinoma of the Liver in 6 Months' Infant

—G. K. Hagaman, St. Paul

Otitis Media in Children—E. J. Huenekens, Minneapolis

Treatment of Enuresis—H. S. Lippman, St. Paul

Intussusception—F. C. Rodda, Minneapolis

Renal Rickets—R. E. Nutting, Duluth

Fecal Incontinence and Megacolon—T. L. Birnberg, St. Paul

Evolution of Tuberculosis in the Human Body—C. A. Stewart, Minneapolis

Case Report, Arteriosclerosis—C. A. Scherer, Duluth

Care of the Premature—A. V. Stoesser, Minneapolis

## Use of Chloral Hydrate in Pediatric Practice—E. D.

Anderson, Minneapolis

Pyelitis—H. F. Helmholz, Rochester

Subacute Atrophy of the Liver—Woodard Colby, St. Paul

Pulmonary Abscess in Children—R. L. J. Kennedy, Rochester

**RADIOLOGICAL SOCIETY**

Room G 14, St. Mary's Hospital—1:30 P. M.

President: Gage Clement Secretary: Leo Rigler  
Radiation Therapy in Non-Malignant Conditions—

Gage Clement, Duluth

Correlative Value of Clinical and Pathological Findings in Roentgenological Diagnosis—Kano Ikeda, St. Paul

The Place of the Roentgenologist in the Private Practice of Medicine—Leo Rigler, Minneapolis

Childhood Tuberculosis—R. G. Allison, Minneapolis

**MINNESOTA ORTHOPEDIC SOCIETY**

Room G 15, St. Mary's Hospital—1:30 P. M.

President: Henry W. Meyerding

Secretary: John Moe

Fractures in and near Joints—E. S. Geist, Minneapolis

Bone Tuberculosis—C. K. Petter, Oak Terrace

Demonstration of Improved Clavicle Splint—M. O.

Henry, Minneapolis

Old and Recent Fractures of Ankle—M. S. Henderson, Rochester

Rôle of the Soft Tissues in the Diagnosis and Treatment of Back Injuries—E. T. Evans, Minneapolis

Fractures of Humerus and Volkmann's Contracture—

Henry W. Meyerding, Rochester

Stiff and Painful Shoulder—J. R. Kuth, Duluth

The Operative Treatment of Congenital Club Feet—

G. A. Williamson, St. Paul

Extra-articular Fusion of the Hip Joint—W. H. Von der Weyer, St. Paul

**SOCIETY OF NEUROLOGY AND PSYCHIATRY**

Auditorium, Nurses' Home—3:45 P. M.

President: R. S. Ahrens Secretary: G. R. Kamman

Psychoneurosis—F. W. Whitmore, St. Paul

General Paresis, W. P. Gardner, St. Paul

The Pre-Senium—G. N. Ruhberg, St. Paul

Spinal Fluid Examinations—A. S. Hamilton, Minneapolis

The Operability of Spinal Cord Tumors—W. McK. Craig, Rochester

Gait Disturbances Due to Neurological Diseases—Moving Pictures—L. R. Gowan, Duluth

**ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY**

East Hall, Nurses' Home—1:30 P. M.

President: H. J. Rothschild Secretary: W. D. Camp

Program presented by sections on Ophthalmology, Otolaryngology, Oral and Plastic Surgery

of The Mayo Clinic

Osteitis Deformans of the Bones of the Face—

F. Z. Havens

Sarcoma of the Larynx—F. A. Figi  
Permanent Enlargement of the Lips and Face,  
Secondary to Recurring Swellings and Associated  
with Facial Paralysis; a Clinical Entity—Gordon  
B. New

#### SYMPOSIUM ON CHRONIC SUPPURATIVE OTITIS MEDIA

Clinical Classification—W. B. Stark  
Conservative Management—C. M. Anderson  
Indications for Surgical Interference—H. I. Lillie  
Operative Procedures—B. E. Hempstead  
**SYMPOSIUM ON GLAUCOMA**  
Changes in Vision and Visual Fields—W. I. Lillie  
Differential Ophthalmoscopic Diagnosis—H. P.  
Wagener  
Non-surgical Management—A. D. Prangen  
Surgical Treatment of Diseases of the Orbit—W. L.  
Benedict

#### Monday Evening, May 22, 1933

##### MEDICAL ECONOMICS MEETING Methodist Church—8 P. M.

N. O. Pearce, Chairman

Address—A. W. Adson, President Minnesota State  
Board of Medical Examiners, Rochester  
Trend of Medical Practice—Morris Fishbein, Chi-  
cago; C. H. Mayo, Rochester  
Remarks by the Legislative Committee—H. M. John-  
son, Chairman, Dawson

#### Tuesday Morning, May 23, 1933

##### GENERAL ASSEMBLY Auditorium—8:30 A. M.

*Presiding Officers:*

A. T. Laird, Nopeming E. M. Hammes, St. Paul  
**SYMPOSIUM ON NUTRITIONAL DISORDERS**  
The Rôle of Vitamins in Nutrition—G. O. Burr,  
Ph.D., Associate Professor of Botany, University  
of Minnesota  
Skin Manifestations of Avitaminosis—S. E. Sweitzer,  
Minneapolis  
Eye Manifestations of Avitaminosis—D. L. Tilder-  
quist, Duluth  
Systemic Manifestations of Avitaminosis—Harry  
Oerting, St. Paul  
Treatment of Malnutrition in Adults—Moses Barron,  
Minneapolis  
Vitamin Deficiency in Children—Philip C. Jeans,  
Professor of Pediatrics, University of Iowa, Iowa  
City  
Summary—W. C. Alvarez, Rochester

11:30 A. M.

Undulant Fever—Walter M. Simpson, Director,  
Diagnostic Laboratories, Miami Valley Hospital;  
President, American Society of Clinical Patholo-  
gists, Dayton, Ohio

#### TABLE DEMONSTRATIONS

East Hall—10:30 A. M.

Practical Points in the Treatment of Rectal Dis-  
orders—L. A. Buie, Rochester  
Animated Drawings of the Origin and Surgical Manage-  
ment of Anal Fistula

Causes of Hay Fever in Minnesota—R. V. Ellis and  
C. O. Rosendahl, Ph.D., Minneapolis

Demonstration and exhibit by the Medical School and  
Department of Botany, University of Minnesota  
Amphitheater, 6th Floor, St. Mary's Hospital

Methods of Anesthesia, including Spinal Anesthesia  
—J. S. Lundy, Rochester

Auditorium Stage

Physiotherapy Demonstration—Arthritis and Acute  
Injuries—A. U. Desjardins, P. S. Hench, and Ann  
Kelley, R.N., Rochester

West Hall—B-24

Complete Medical and Laboratory Examination of  
a Patient—W. C. Alvarez, Rochester

West Hall—B-27

Practical Application of Intravenous Urography—  
William F. Braasch, Rochester

West Hall—B-20

Treatment and Complications of Gonorrhea—A. L.  
Clark, Rochester

Demonstrating the diagnostic technic, including staining of  
slides and irrigation of patient

West Hall—B-22

Exhibit on Radiotherapy, Roentgen Rays and Radium  
—A. U. Desjardins and H. H. Bowing, Rochester  
Presented by Section of Therapeutic Radiology, Mayo Clinic

West Hall—Booth No. 24

Treatment of Acute Burns—R. K. Ghormley and  
E. G. Bannick, Rochester

Demonstration and Exhibit showing enlarged photographs,  
charts and equipment.

West Hall—Booths No. 21 and 22

The Occurrence of Tularemia in Man and Animals—  
R. G. Green, University of Minnesota

West Hall—Booths No. 7 and 8

Professional Pharmacy as Applied to the Physicians  
—Under the auspices of the Inter-Professional  
Relationships Committee of Hennepin County

West Hall—B-26

Liver Function Tests—T. B. Magath, Rochester

West Hall—B-28

Treatment of Syphilis—P. A. O'Leary and L. A.  
Brunsting, Rochester

West Hall—Booth No. 27

(1) The Geiger Counter and Its Application to the  
Measurement of the Circulation Time of the  
Blood; (2) The Electromotive Thermometer and  
the Measurement of Skin Temperature; (3) Short  
Electric Wave (Radiotherapy) Apparatus—Charles  
Sheard, Ph.D., Rochester

(This apparatus will be demonstrated to those who make  
application at the Registration Desk before 9 o'clock Monday  
and Tuesday)

West Hall—Booth No. 6

Minnesota General Hospital—Relationship to the  
Medical Profession

#### Tuesday Afternoon, May 23, 1933

##### GENERAL ASSEMBLY

Auditorium—1:45 P. M.

*Presiding Officers:*

A. T. Laird, Nopeming J. C. Masson, Rochester  
CLINICS

Dislocations—Dean Lewis, Professor of Surgery,  
Johns Hopkins University School of Medicine;

## PROGRAM EIGHTIETH ANNUAL MEETING

351

President, American Medical Association, Baltimore  
Surgery—E. S. Judd, Rochester

Urology—H. L. Kretschmer, Clinical Professor of Surgery, Rush Medical College, Chicago

Pediatrics—Irvine McQuarrie, Minneapolis

Neuro-Surgery—A. W. Adson, Rochester

Medicine—Frank J. Hirschboeck, Duluth

## SCIENTIFIC CINEMA

Room G 15—8:45 A. M.

J. G. Mayo, Chairman

L. A. Julin, Technical Supervisor

M. W. Else, Projection Supervisor

Thyroidectomy—C. F. Dixon, Rochester

Cardiac, Vasomotor, and Respiratory Phenomena with an Analysis of the Signs and Symptoms of Experimentally Raised Intracranial Pressure—(Talkie) Courtesy of Petrolagar Company

Teaching Film for Undergraduate Neurology—J. C. McKinley, Lecturer and Producer, University of Minnesota

Anatomy of the Female Pelvis and Perineum—(Talkie) Courtesy of Petrolagar Company

Mayo Vaginal Hysterectomy and Perineorrhaphy—V. S. Counsellor, Lecturer and Producer, Rochester

Roentgeno-cinematic Pictures of the Movements of the Stomach—Cesare Gianturco, and W. C. Alvarez, Lecturer and Producer, Rochester

Increased Frequency of Winking and the Contraction of the Orbicularis Oculi and Associated Facial Muscles in Exophthalmic Goiter; Motion Picture Demonstration—W. A. Plummer and E. H. Ryneerson, Lecturers and Producers, Rochester

Living Sutures in Repair of Hernia—J. C. Masson, Lecturer and Producer, Rochester

## SCIENTIFIC CINEMA

Room G 15—1:30 P. M.

Vaginal Hysterectomy for Uterine Prolapse—(Talkie) Courtesy of Petrolagar Company

A Practical Method of Filming Surgical Procedure Available to Everyone—L. A. Julin, Lecturer and Producer, Rochester

(1) Posterior Polya Resection of the Stomach for Benign and Malignant Lesions; (2) Billroth No. 1 for Gastric and Duodenal Lesions—Waltman Walters, Lecturer and Producer, Rochester

Eye Operations. Cataract; Glaucoma; Enucleation with Fat Implant; Magnet Operation, etc.—C. N. Spratt, Lecturer and Producer, Minneapolis

(1) Intratracheal Anesthesia; (2) Sacral Block Anesthesia, Part II; (3) Pulmonary Ventilator; (4) Spinal Anesthesia—J. S. Lundy, Lecturer and Producer, Rochester

Traumatic Surgery of the Extremities—H. W. Meyerding and R. K. Ghormley, Lecturers, Rochester; Davis & Geck, Producers, Brooklyn, N. Y.

**Tuesday Evening, May 23, 1933**

Methodist Church—8:00 P. M.

*Presiding Officer:* W. J. Mayo

Address of Welcome—G. B. Eusterman, President Olmsted-Houston-Fillmore County Medical Society, Rochester

President's Address—N. O. Pearce, Minneapolis

Address by President-Elect

The Women's Auxiliary—Mrs. A. A. Passer, President-elect, Olivia

The Medical Profession versus Racketeering—His Excellency, John Gregory Murray, Archbishop of the Diocese of St. Paul

Address—Dean Lewis, President American Medical Association, Baltimore

9:00 P. M.—Reception—Mayo Clinic

**Wednesday Morning, May 24, 1933**

## GENERAL ASSEMBLY

Auditorium—8:30 A. M.

*Presiding Officers:*

J. C. Masson, Rochester    R. E. Swanson, Minneapolis

## SYMPOSIUM ON EMERGENCY SURGERY

Farm Accidents—M. O. Oppgaard, Crookston

Auto Accidents—Kenneth Bulkley, Minneapolis

Industrial Accidents—Paul H. Kelly, St. Paul

Emergency Surgery of the Abdomen—M. G. Gillespie, Duluth

Emergency Surgery in Infants and Children—O. S. Wyatt, Minneapolis

Strangulated Hernia—George A. Geist, St. Paul

Presentation of Officers

Citizens Aid Society Memorial Address—James Ewing, Director, Memorial Hospital, Professor of Oncology, Cornell Medical School, New York

Do's and Don'ts of the Injection of Varicose Veins—H. O. McPhee, Minneapolis

Over 5,000 Mantoux Tests in Polk and Norman Counties—W. G. Paradis and O. E. Locken, Crookston

## SCIENTIFIC CINEMA

Room G 15—9:00 A. M.

Cardiac, Vasomotor, and Respiratory Phenomena with an Analysis of the Signs and Symptoms of Experimentally Raised Intracranial Pressure—(Talkie) Courtesy of Petrolagar Company

Roentgeno-cinematic Pictures of the Movements of the Stomach—W. C. Alvarez and Cesare Gianturco, Lecturers and Producers, Rochester

Living Sutures in Repair of Hernia—J. C. Masson, Lecturer and Producer, Rochester

Mayo Vaginal Hysterectomy and Perineorrhaphy—V. S. Counsellor, Lecturer and Producer, Rochester

(1) Posterior Polya Resection of the Stomach for Benign and Malignant Lesions; (2) Billroth No. 1 for Gastric and Duodenal Lesion—Waltman Walters, Lecturer and Producer, Rochester

Traumatic Surgery of the Extremities—H. W. Meyerding and R. K. Ghormley, Lecturers, Rochester; Davis & Geck, Producers, Brooklyn, New York

## TABLE DEMONSTRATIONS

East Hall—11:15 A. M.

Practical Points in the Treatment of Rectal Disorders—L. A. Buie, Rochester

Animated Drawings of the Origin and Surgical Management of Anal Fistula

X-rays Illustrating Reaction of Human Gall Bladder to Faradic Stimulation of Stomach—E. A. Boyden

Emptying of the gall bladder in children

Amphitheater, 6th Floor, St. Mary's Hospital

Methods of Anesthesia, including Spinal Anesthesia—J. S. Lundy, Rochester

Auditorium Stage

Physiotherapy Demonstration—Arthritis and Acute Injuries—A. U. Desjardins, P. S. Hench, and Ann Kelley, R.N., Rochester

West Hall—B-24

Complete Medical and Laboratory Examination of a Patient—W. C. Alvarez, Rochester

West Hall—B-20

Treatment and Complications of Gonorrhea—A. L. Clark, Rochester

Demonstrating the diagnostic technic, including staining of slides and irrigation of patient

West Hall—B-22

Exhibit on Radiotherapy, Roentgen Rays and Radium—A. U. Desjardins and H. H. Bowing, Rochester

Presented by Section of Therapeutic Radiology, Mayo Clinic

West Hall—Booth No. 24

Treatment of Acute Burns—R. K. Ghormley and E. G. Bannick, Rochester

Demonstration and Exhibit showing enlarged photographs, charts and equipment

West Hall—Booths No. 21 and 22

The Occurrence of Tularemia in Man and Animals—R. G. Green, University of Minnesota

West Hall—Booths No. 7 and 8

Professional Pharmacy as Applied to the Physicians—Under the auspices of the Inter-Professional Relationships Committee of Hennepin County

West Hall—B-27

A Study of Hypertension—N. M. Keith, H. P. Wagner and N. W. Barker, Rochester

Clinical Groups, Ophthalmoscopic Findings, Pathologic Changes in Arterioles

West Hall—B-26

Studies in Suprarenal Physiology—E. C. Kendall, Rochester

West Hall—B-28

Treatment of Syphilis—P. A. O'Leary and L. A. Brunsting, Rochester

West Hall—Booth No. 6

Minnesota General Hospital—Relationship to the Medical Profession

Luncheon Recess—12:15 P. M.

Golf Tournament—Country Club—2:00 P. M.

## Scientific Exhibits

East Hall

Cancer Control in Minnesota—American Society for the Control of Cancer

Charts of distribution of cancer mortality in Minnesota and United States. Moulages of breast cancer. Photos of transillumination of breast tissue. Motion picture of growth of normal and sarcoma tissue and the effect of radium emanation on both types of tissue. Literature on cancer for physicians.

Oleothorax in Pulmonary Tuberculosis—H. Longstreet Taylor Foundation, Pokegama Sanatorium

Tuberculosis as Disclosed by X-ray of the Chest—F. E. Harrington, Minneapolis, and E. S. Mariette, Oak Terrace

108 selected x-ray transparencies of the chests of children and adults, showing demonstrable tuberculosis

Undulant Fever—Minnesota Department of Health, Division of Preventable Diseases, Minneapolis

Urethrography—M. A. Nicholson and M. J. Fiala, Duluth

X-rays of urethra showing normal and pathologic urethras. Methods used in making films. Media and special apparatus

Over 5,000 Mantoux Tests in Polk and Norman Counties—W. G. Paradis and O. E. Locken, Crookston

West Hall—Booths No. 7 and 8

Professional Pharmacy as Applied to the Physicians—Under the auspices of the Inter-Professional Relationships Committee of Hennepin County

Booth No. 15

Minnesota Public Health Association

Booth No. 14

Minnesota State Medical Association

West Hall—Booth No. 5

Minnesota State Board of Medical Examiners

Booth to be assigned

Women's Auxiliary, American Medical Association

East Hall

## THE MAYO FOUNDATION FOR MEDICAL EDUCATION AND RESEARCH

Experimentally Produced Peptic Ulcer—F. C. Mann and J. L. Bollman

Leiomyomata of the Stomach—J. H. Rieniets

Studies of Gastric Secretion—M. W. Comfort

Gastric Syphilis—G. B. Eusterman

The Significance of Hematemesis—A. B. Rivers

Ulcers of the Duodenum—H. E. Robertson

Roentgenograms of Stomach and Duodenum—B. R. Kirklin

Lesions of the Stomach—D. C. Balfour and H. I. Down

Relation of Streptococci to the Etiology of Ulcer—E. C. Rosenow

Carcinoma of the Bronchus—H. J. Moersch and P. P. Vinson

Roentgenologic Study of Interesting Lesions in Childhood—R. L. J. Kennedy and J. D. Camp

The Management of Permanent Colostomies and Ileostomies—J. A. Bargen

Pathologic Conditions of the Jaws—Gordon B. New, F. A. Fagi and H. F. Havens

## UNIVERSITY OF MINNESOTA

Causes of Hay Fever in Minnesota—R. V. Ellis and C. O. Rosendahl, Ph.D.

Exhibit by the Medical School and Department of Botany, University of Minnesota

West Hall—Booth No. 6

Minnesota General Hospital—Relationship to the Medical Profession

West Hall—Booths No. 21 and 22

## The Occurrence of Tularemia in Man and Animals—

R. G. Green

Charts of occurrence; demonstration of pathological material; cultures; agglutination tests; skin tests; demonstrations of insect vectors and animal hosts

## WOMAN'S AUXILIARY

President—MRS. EDWARD SCHONS, Saint Paul  
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Minneapolis

Editor—MRS. S. H. BAXTER, Minneapolis

Plans will be completed soon for one of the pleasantest and the most profitable annual meetings in the history of the State Auxiliary. It is to be held at Rochester May 22, 23 and 24 in conjunction, as always, with the State Meeting of the Minnesota State Medical Association.

The outstanding event of the three days for the women will undoubtedly be the annual luncheon, Wednesday, May 24, at which Mrs. James S. Percy of Los Angeles, president of the Auxiliary to the American Medical Association, will be present as guest of honor. Sharing honors will be Mrs. James Blake, our Minnesota president-elect of the National Auxiliary, who succeeds Mrs. Percy. The occasion will be unique in the annals of the State Auxiliary.

Programs with complete details of the meetings and of the social events planned by the hostess organization, the Auxiliary to the Olmsted-Houston-Fillmore County Medical Society, will be mailed to all doctors' wives several days before the opening of the meeting.

It is to be hoped that every doctor's wife, whether or not she is a member of the Auxiliary, will attend. The meeting affords extraordinary opportunities to attend many of the distinguished scientific events arranged for the physicians, also to visit the famous libraries and museums of Rochester and to participate in a series of teas and receptions in charming Rochester homes.

(MRS. S. H.) LAURA R. BAXTER.

## PROCEEDINGS OF THE MINNESOTA ACADEMY OF MEDICINE

Meeting of March 8, 1933

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, March 8, 1933. Dinner was served at 7 p. m. and the meeting was called to order at 8 p. m. by the President, Dr. C. D. Freeman. There were 55 members and 1 visitor present.

Minutes of the February meeting were read and approved.

Dr. A. S. Hamilton read the following memorial to Dr. Thomas G. Lee; and a motion was carried that it be spread on the minutes of the Academy and a copy sent to the family.

Dr. THOMAS G. LEE, the last member of the original faculty of the College of Medicine of the University of Minnesota to retire from teaching service, died September 1, 1932, from injuries received in an automobile accident four days previously.

Dr. Lee was born in 1860 at Jacksonville, New York. He was graduated from the University of Pennsylvania in 1886 and held the degrees of Bachelor of Science and Doctor of Medicine from that school and, later, of Bachelor of Science from Harvard. For five years he was lecturer in Histology and Embryology at Yale University and also taught the same subjects at Radcliffe College for one year.

When the Medical School of the University of Minnesota was organized, Dr. Lee, in 1892, was given charge of the laboratory of Histology and Embryology, and also of Bacteriology and Clinical Microscopy. The latter subjects were soon turned over to other men and Dr. Lee continued as Professor in Charge of Histology and Embryology. In 1908 he became Professor and head of the Department of Anatomy and in 1913 he was made Professor of Comparative Anatomy and continued in this position until his retirement from active teaching in 1929 when he went to Florida to spend the remainder of his days.

When Dr. Lee came to the University, the Medical Department was limited in attendance and in quarters. With Dean Wulling he shared a one-story frame building, and the building that was to house the medical, dental and pharmacy departments was then in the process of erection. The medical course covered two years only.

Dr. Lee saw the Campus, and especially the medical

Campus, steadily expand. He visited Europe in connection with the plans for a new Institute of Anatomy, which was completed in 1913. For years Dr. Lee was Secretary of the Medical Faculty and was Librarian of the Medical School. He taught with or under each of the University's presidents. All of his life was devoted to administration, teaching and research. He was an indefatigable worker, was especially interested in the embryology of certain mammals, published several papers on that subject, in which he was a recognized authority, and left a valuable collection behind him. During his lifetime he was a member of many scientific bodies, including the American Association for the Advancement of Science, Sigma XI, American Society of Naturalists, American Society of Zoology, American Medical Association, Minnesota Academy of Medicine, American Association of Anatomists and Anatomische Gesellschaft. He was also deeply interested in Masonry, was a Charter Member of the Acacia Masonic fraternity and was second Master of the University Lodge of the Masonic order as well as a charter member. He was President of the Council of Knights Templar of the Masonic order, as well as a charter member and a 33rd degree Mason.

Dr. Lee was a very modest man, taking little part in public life. He was a widely read man with a special knowledge of comparative and ancient religions. He enjoyed the loyalty and the affection of his colleagues and of his students. On December 21, 1887, he was married to Miss Emma Louise Shaw, of West Bloomfield, New York. Though no longer a member of our Medical Faculty, regard for his memory is joined with deep sympathy for his wife, who survives him.

(Signed) The Committee:

ARTHUR S. HAMILTON, Chairman,  
F. R. WRIGHT,  
S. MARX WHITE.

Upon ballot, the following men were elected to membership in the Academy: Dr. B. S. Adams (Associate) of Hibbing; Dr. H. W. Cook (Active) of Minneapolis; and Dr. H. L. Dunn (University) of Minneapolis.

The scientific program of the evening consisted of two papers.

## TRICHOMONAS VAGINALIS VAGINITIS

W. H. CONDIT, M.D., Minneapolis

## ABSTRACT

It is with apology that I offer these remarks on a subject so generally discussed in recent medical literature, but I have three reasons for so doing.

First, all members of the medical profession, not excepting some with a dental degree, treat the vaginal outlet therapeutically.

Second, there are not a few in our profession who do not recognize trichomonas vaginitis as a definite entity.

Third, it was my good fortune to have access to all publications by Dr. Hegner, Professor of Parasitology at Johns Hopkins University, who has done most extensive study on the Trichomonads the past six years.

Most gynecologists now recognize the condition known as Trichomonas vaginalis vaginitis as a definite entity. My interest was aroused some two years ago in an endeavor to determine why the profession has not recognized the Trichomonads present in vaginal discharges as a primary or secondary etiological factor in the one hundred years that have elapsed since the first discovery of their presence in the vagina by Donné.

The first clinical report in American literature on the treatment of vaginal discharge associated with the presence of Trichomonads was presented by DeLee in the Illinois State Medical Journal in 1920. The second report was by Greenhill before the Chicago Gynecological Society in 1928, published in the *Journal of the American Medical Association*, May 3, 1931. This last report brought many inquiries from the profession, including many gynecologists who had not recognized a single case. Mathieu, Carl Davis, N. S. Davis, Singleton and others have contributed to the literature on this subject.

*Conclusions:* (1) Trichomonas vaginalis vaginitis, if not proven, is an accepted entity.

(2) Diagnosis and treatment of the condition will result in the cure of many annoying symptoms accompanying the vaginitis and assist in lowering puerperal morbidity.

(3) A standard treatment has not been established but recommended procedures are sufficient to yield gratifying results.

## DISCUSSION

DR. F. R. WRIGHT (Minneapolis): There were two things in this paper which interested me. One is the statement that this germ has been found in prostatic secretion. Now prostatic secretion is obtained by holding the meatus, massaging the prostate and collecting the secretion from the urethra. Therefore, you can't tell whether bacteria in the secretion are from the prostate or from the urethra. Some dozen bacteria are found near the meatus; as you go back farther in the urethra there are fewer, and the posterior is practically always sterile. So that, if bacteria are found in prostatic secretion, there is no way of telling whether they are from the prostate or the urethra.

The other point is about the use of soap. Soap is a solvent. When it is put into the vagina it washes out the secretions. The soap which Dr. Condit speaks of is a soft soap and not purified. Ordinary green soap contains an excess of alkali. It may be possible that, in washing out the vagina with this soap, it is the excess of alkali in the soap that does the work.

DR. J. L. ROTHRICK (St. Paul): The rôle of the Trichomonas vaginalis in the pathology of vaginitis is still undecided. Until comparatively recently it was considered a harmless organism which was frequently found in the vagina of women.

It was not until 1916 that Höhne ascribed to it the rôle of an important etiological factor of vaginitis. He believed that it was not only the sole cause of many cases of vaginitis but that, instead of its being a harmless parasite, it frequently invaded the deeper struc-

tures and was responsible at times for vulvo-vaginal abscesses and other lesions beneath the superficial layers of the epithelium of the vagina; a position from which he was later obliged to recede.

In the years that followed, numerous papers appeared in German literature and opinion was pretty evenly divided. On account of the embargo on German publications occasioned by the war, these did not become accessible to American readers until about 1920. Since then this subject has been much discussed in American literature.

It is important that we keep in mind a few of its characteristic features. While it is possible to grow it on artificial media, no one has as yet been able to procure it in pure culture.

Inoculation of the vagina of healthy women with the organism grown on artificial media or even with vaginal secretion obtained from a case of vaginitis, fails to produce the disease.

It is frequently found in the vagina of women who show no sign of vaginitis.

It is limited in its distribution to the vaginal tract and vulva, involvement of the external genitalia being probably made possible by irritation from the discharge.

It does not invade the uterine cavity.

There is no specific treatment but it yields promptly to various antiseptics.

Are we justified, therefore, in speaking of a trichomonas vaginitis?

The pronounced tendency to recurrence, which has been emphasized as a proof of its specific etiology may possibly be partly explained by a lowered resistance in certain individuals on the ground of constitutional disease with the disturbance of the balance of the endocrine glands just as we recognize that the invasion of the vagina by pathogenic bacteria is favored by such disturbance.

In point of its clinical importance it is interesting to call to mind the frequency of vaginitis in pregnant women with its possible bearing in the production of puerperal morbidity. The weight of opinion today is that it does not tend materially to increase it. Nevertheless it is important that vaginitis occurring in pregnant women should be promptly treated.

If this discussion has accomplished no other result it has focused attention on the importance of rational treatment as applied to vaginitis, which I fear has fallen into neglect in recent years.

DR. CONDIT (in closing): I was very much interested in Dr. Rothrock's remarks relative to lowered resistance in the female being a predisposing factor in the occurrence of vaginitis. MINNESOTA MEDICINE of April, 1931, published a paper on non-specific vaginitis which I had presented before the annual meeting of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons at Niagara Falls, Ontario, September, 1930, in which I laid great stress upon this point of lowered resistance. I can recall many patients suffering recurrences of vaginal discharges on the occurrence of severe or mild infections, physical tire, or worry. We do find this flagellate in many human mouths and vaginas of patients in whom no symptoms of their presence or activity occur.

NOTE: Since the meeting of March 8, I have received correspondence from Robert Hegner, Ph.D., Professor of Protozoölogy, Johns Hopkins University, which I offer here as a supplement to this discussion:

March 29, 1933.

Dr. William H. Condit,  
1009 Nicollet Avenue,  
Minneapolis, Minn.

Dear Dr. Condit:

I do not believe it is yet certain that Trichomonas vaginalis causes inflammation or vaginitis. Pure cultures of Trichomonas vaginalis have been grown many times. A correspondent from one of the Southern States claims to have brought about vagi-

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nitis in colored women by inoculating them with the flagellate, but he certainly included bacteria along with the trichomonads. I am not quite sure of your definition of a pure culture. Trichomonads do not appear to grow in culture medium in the absence of bacteria. As you state, the general impression is that *T. vaginalis* is pathogenic, and I am inclined to this opinion myself, but it certainly has not been definitely proven. I have recently obtained vaginal infections in monkeys with *T. hominis* from the intestine of man, but these infections seem to be temporary. I do not believe my previously published results prove anything more than that.

Very sincerely yours,

(Signed) ROBERT HEGNER

#### POSTOPERATIVE PERITONEAL ADHESSIONS

##### Some Causes and Means of Prevention

A. E. BENJAMIN, M.D., Minneapolis

Paper with discussions by Drs. A. A. Zierold and O. H. Wangenstein is to be published at a later date in MINNESOTA MEDICINE.

R. T. LA VAKE, M.D., Secretary.

## BOOK REVIEWS

Books listed here become the property of the Ramsey and Hennepin County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

**TEN YEARS OF OBSTETRICS AND GYNECOLOGY IN PRIVATE PRACTICE.** John L. Rothrock, A.B., M.D., F.A.C.S. Formerly Associate Professor of Obstetrics and Gynecology, University of Minnesota; former member Miller Clinic, and Chief of Obstetrical and Gynecological Services of Charles T. Miller Hospital and Wilder Dispensary, Saint Paul, Minnesota. 209 pages. Illus. Price, \$3.50, cloth. New York: Paul B. Hoeber, Inc., 1933.

**CHININUM IN DER ALLGEMEINPRAXIS.** Dr. Med. Fritz Johannesohn, of Mannheim. 175 pages. Amsterdam, Holland: Bureau for Increasing the Use of Quinine, 1933.

**CHRONIC ARTHRITIS AND FIBROSITIS: DIAGNOSIS AND TREATMENT.** Bernard Langdon Wyatt, M.D. 201 pages. Illus. Price, \$3.50. Baltimore: William Wood & Co., 1933.

A better title for this monograph might have been "Recent Advances in Chronic Arthritis," as the book is more or less of a review of the recent work done in this field. The author quotes at length from the most recent research done in the field of chronic rheumatism. However, this book is of considerable interest to one concerned with this problem, as it gives a clear-cut, conservative point of view of the entire subject. This particular field is so confused and so controversial that it is especially difficult to give an unbiased review. The author has accomplished this in excellent fashion and he has been able to write a brief and easily understood résumé.

The first four chapters consider the statistical aspect, especially the nomenclature, pathology and differential diagnosis. The author has accepted the classification provided by the American Committee for the Control of Rheumatism but he has unfortunately elected to use synonymously the term, chronic infectious arthritis with

proliferative arthritis. Because of the controversy which is now going on as to the etiology of chronic arthritis it would seem better to use the term, chronic infectious arthritis only for those cases in which the specific infection is known. The use of the term, infectious arthritis indicates that other types of arthritis are noninfectious. Some of the most recent work would tend to indicate that all types of arthritis may be of bacteriological origin. It appears that all types of arthritis can be grouped under the two headings, proliferative and degenerative arthritis.

The fourth and fifth chapters consider therapeutic measures. The author discusses not only the general treatment necessary, such as the removal of the foci of infection and methods of increasing the resistance of the patient, but also gives detailed information in the use of physio-therapeutic measures. The value of massage, heat and occupational therapy is stressed and the point is correctly brought out that physiotherapy is sadly neglected in this country.

The author has accepted as an additional method of treatment, the use of intravenous vaccine and states that he has obtained a good percentage of improvement with the use of intravenous streptococcus vaccine. One would like to know on what grounds this conclusion was made when it is considered that various methods of treatment were used at the same time. It seems a difficult task to determine exactly how much value was actually obtained by the use of vaccine injections without the use of control experiments.

This book can be well recommended to any one interested in this subject as it gives a well written and comprehensive study of the recent work on arthritis.

M. J. SHAPIRO, M.D.

**THE TECHNIC OF CONTRACEPTION.** Eric M. Matsner, M. D. Published for the American Birth Control League by the Williams & Wilkins Company of Baltimore, 1933.

The pamphlet contains illustrated directions for using various mechanical devices for the prevention of conception. It has to admit that ideal prevention has not yet been discovered, and that it has been impossible to find an infallible method to circumvent nature's aim to reproduce species.

WILLIAM DAVIS, M.D.

**THE PRACTICAL MEDICINE SERIES, GENERAL MEDICINE.** By a corps of editors: Series 1932. Chicago: The Year Book Publishers, Inc., 1932.

It is a surprise to find arthritis included in the section of infectious diseases with which this volume begins. Infectious diseases and contagious diseases have never been separated from one another satisfactorily nor their limits defined; still it is unexpected to find arthritis grouped with diphtheria, measles, smallpox and other diseases communicable by direct or indirect contact and arising only from cases of the same kind. In this section considerable space is given to the comparatively new diseases, psittacosis, tularemia, and undulant fever, all of which are on the increase in this country and now are found distributed quite generally.

Under smallpox is given an abstract of a paper by Woodward in the *New England Journal of Medicine* for March 17, 1932, containing new figures upon vaccination. Four states have laws against compulsory vaccination. These "free" states, so-called by the anti-vaccinationists, are Arizona, Utah, North Dakota, and Minnesota, and in 1920 their combined population was 35,000 less than that of Massachusetts. In the ten years 1919-1928 these "free" states had a total of 46,130 cases of smallpox, while in Massachusetts there were but 408. What a glorious company Minnesota is in!

To protect themselves against closure for quarantine because of smallpox, twelve of the leading preparatory schools and thirty-six of the foremost universities and colleges, both for men and for women, now admit no unvaccinated pupils.

The section on diseases of the chest, excepting the heart, is by Lawrason Brown, of Trudeau Sanatorium, and naturally tuberculosis has the first place. Much is said of x-ray diagnosis, B C G vaccination, trauma, and the surgery of the thorax. Minnesota figures largely in this section, with papers from Pokegama on the use of insulin to improve the appetite, on the results in control by Meyerding, on the detection of the disease in a farming community, by Simmons, and again by Anderson, on the prevalence of tuberculosis among students at the University by Wulff and Myers, its control by Diehl, its prevalence among nurses by Geer, and its management by surgery by Wangenstein.

In the section on diseases of the blood and blood-forming organs, edited by Minot and Castle, of Boston, it is not surprising that pernicious anemia holds the most prominent place. Stress is laid upon the necessity of keeping up the liver treatment and of attention to the general diet, giving digestible foods with an abundance of fruit and green vegetables. To fix an amount of liver that will apply to all cases would be as futile as to try to establish a dose of insulin that would suit every diabetic.

William D. Stronk, of the University of Pennsylvania, has the section on diseases of the heart and blood vessels. Here again is an extensive quotation from an important paper by a Minnesota doctor, a review of subacute bacterial endocarditis by S. Marx White, of Minneapolis. This disease is so closely associated with Osler, who wrote a classical description of it, that it might fairly be called Osler's disease, a name that is indeed not infrequently applied to it. It is encouraging to learn that White finds it much less hopeless than was thought formerly, although he has no specific to offer for it.

Under the heading "Diseases of the Digestive System and Metabolism," managed by Ralph C. Brown, of Rush, there is an abstract of a good paper on duodenal ulcer by John A. Ryle, published in the *London Lancet* last year, and urging that surgeons and physicians keep in mind ulcer the disease and not merely the local lesion. In other words the patient should be treated in mind and body and correction should be made of bad habits such as irregular and bolted meals, excessive smoking, and the intrusion of business and other worries upon the digestion. There are interesting observations by several other writers upon various aspects of the problems both of duodenal and of gastric ulcers.

WILLIAM DAVIS, M.D.

#### HEALTH ON THE FARM AND IN THE VILLAGE. D. E. A. Winslow, Dr.P.H. 281 pages. Illus. Price \$1.00. New York: The Macmillan Co., 1931.

This volume is an evaluation of work done in a health demonstration in a county of 72,000 (Cattaraugus County, New York). The work was initiated by Milbank Fund, which supplied a large part of the money at the beginning of the demonstration. At the end of seven years (1929) Dr. Winslow and a corps of workers were called in to evaluate in an impartial way the work accomplished.

The dictum of Dr. Biggs "Public Health is Purchasable" was again exemplified by this demonstration. There was a marked reduction in diphtheria, tuberculosis and infant mortality, all of which were reduced below the normal reductions.

Another big achievement was the convincing of the county authorities of the value of the demonstration and thus winning their financial support. The cost in 1929 was \$3.39 per capita. The book is primarily of interest to Public Health Workers.

F. L. JENNINGS, M.D.

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Secretary		Halpin, J. E.	Rush City	Phelps, A. G.	St. Paul
Schoofs, G. E.	North Branch	Hedenstrom, L. H.	Cambridge	Roehlke, A. B.	Elk River
		Heseltine, V. G.	Taylors Falls	Schoofs, G. E.	North Branch
Arends, A. L.	Askov	Holmes, A. E.	Rush City	Stephan, E. L.	Hinckley
Blumenthal, J. S.	Columbia Heights	Johnson, C. E.	Cambridge	Stratte, A. K.	Pine City
Brownstone, Manuel	Sandstone	Kelsey, C. G.	Hinckley	Swenson, Charles	Braham
Callahan, F. F.	Pokegama	Kooiker, H. I.	Milaca	Truog, C. P.	Lindstrom
Cooney, H. C.	Princeton	McBroom, D. E.	Cambridge	Vik, Melvin	Onamia
Drege, H. P.	Hinckley	Nethercott, E. G.	Pine City	Vrooman, F. E.	St. Francis
		Norman, W. F.	Mora	Zeien, Thomas	North Branch

[May, 1933]

## FREEBORN COUNTY MEDICAL SOCIETY

Regular meetings, quarterly  
Annual meeting, December

President	
Burns, H. D.	Albert Lea
Secretary	
Freigh, W. P.	Albert Lea
Branham, D. S.	Albert Lea
Burns, H. D.	Albert Lea

Butturff, C. R.	Freeborn
Calhoun, F. W.	Albert Lea
Folken, F. G.	Albert Lea
Freeman, J. P.	Albert Lea
Freigh, W. P.	Albert Lea
Gamble, J. W.	Albert Lea
Gamble, P. M.	Albert Lea
Gamble, R. M.	Albert Lea

Gullixson, A.	Albert Lea
Kaasa, L. J.	Albert Lea
Kamp, B. A.	Albert Lea
Leopard, B. A.	Albert Lea
Palmer, C. F.	Albert Lea
Palmer, W. L.	Albert Lea
Schultz, J. A.	Albert Lea
Whitson, S. A.	Alden

## GOODHUE COUNTY MEDICAL SOCIETY

Annual meeting, usually December

President	
Brusegard, J. F.	Red Wing
Secretary	
Steffens, L. A.	Red Wing
Aanes, A. M.	Red Wing
Anderson, S. H.	Red Wing

Brusegard, J. F.	Red Wing
Claydon, D. R.	Red Wing
Claydon, H. F.	Zumbrota
Claydon, L. E.	Red Wing
Cremer, M. H.	Red Wing
Cremer, P. H.	Hastings
Fortney, G. O.	Zumbrota
Hedin, R. F.	Red Wing
Johnson, A. E.	Red Wing

Jones, A. W.	Red Wing
Juers, E. H.	Red Wing
Liffrig, W. W.	Goodhue
McGuigan, H. T.	Red Wing
Miller, F. J.	Red Wing
Smith, M. W.	Red Wing
Steffens, L. A.	Red Wing
Vaaler, T.	Cannon Falls
Williams, M. R.	Cannon Falls

## HENNEPIN COUNTY MEDICAL SOCIETY

Regular meetings, first Monday of each month  
Annual meeting, October

President	
Hansen, E. W.	Minneapolis
Secretary	
Campbell, O. J.	Minneapolis
Ahrens, R. S.	Minneapolis
Allen, H. W.	Minneapolis
Allison, R. G.	Minneapolis
Almquist, H. E.	Minneapolis
Altow, H. O.	Minneapolis
Andersen, A. G.	Minneapolis
Andersen, S. C.	Minneapolis
Anderson, D. D.	Minneapolis
Anderson, E. D.	Minneapolis
Anderson, E. R.	Minneapolis
Anderson, F. J.	Minneapolis
Anderson, J. K.	Minneapolis
Anderson, K. W.	Minneapolis
Anderson, P. A.	Minneapolis
Anderson, U. S.	Minneapolis
Andreassen, E. C.	Minneapolis
Annis, H. B.	Minneapolis
Arey, H. C.	Excelsior
Arvidson, C. G.	Minneapolis
Aune, Martin	Minneapolis
Aurand, W. H.	Minneapolis
Avery, J. F.	Minneapolis
Baken, M. P.	Minneapolis
Baker, A. T.	Minneapolis
Baker, E. L.	Minneapolis
Baker, Looe	Minneapolis
Barber, J. P.	Minneapolis
Barron, Moses	Minneapolis
Bas, G. W.	Minneapolis
Baxter, S. H.	Minneapolis
Beard, A. H.	Minneapolis
Beard, R. O.	Minneapolis
Bedford, E. W.	Minneapolis
Bell, E. T.	Minneapolis
Bell, J. W.	Minneapolis
Benedict, E. E.	Minneapolis
Benjamin, A. E.	Minneapolis
Benn, F. G.	Minneapolis
Berkwitz, N. J.	Minneapolis
Bessesen, A. N., Sr.	Minneapolis
Bessesen, A. N., Jr.	Minneapolis
Bessesen, W. A.	Minneapolis
Bessesen, D. H.	Minneapolis
Blake, James	Hopkins
Blautone, H. H.	Minneapolis
Brockman, M. W. H.	Minneapolis
Boies, L. R.	Minneapolis
Booth, A. E.	Minneapolis
Boreen, C. A.	Minneapolis
Borgeson, E. J.	Minneapolis
Bouman, H. A. H.	Minneapolis
Boynton, Ruth	Minneapolis
Bracken, H. M.	Claremont, Calif.
Bratrud, A. F.	Minneapolis
Brown, E. D.	Minneapolis
Brown, E. J.	Minneapolis
Bukley, Kenneth	Minneapolis
Ballard, Mattie J.	Minneapolis
Butler, John	Minneapolis

Buzzelle, L. K.	Minneapolis
Cable, M. L.	Minneapolis
Cahot, G. S.	Minneapolis
Cabot, V. S.	Minneapolis
Cady, L. H.	Minneapolis
Callstrom, G. W.	Minneapolis
Cameron, Isabel	Minneapolis
Camp, W. E.	Minneapolis
Cavanor, F. T.	Minneapolis
Cherry, C. H.	Minneapolis
Chesley, A. J.	Minneapolis
Christenson, C. R.	Minneapolis
Christenson, H. W.	Minneapolis
Clark, H. S.	Minneapolis
Cohen, S. S.	Oak Terrace
Condit, W. H.	Minneapolis
Cook, H. W.	Minneapolis
Cooperman, H. O.	Minneapolis
Corbett, J. F.	Minneapolis
Corniea, A. D.	Minneapolis
Cosman, E. O.	Minneapolis
Cottam, G. G.	Minneapolis
Crafts, L. M.	Minneapolis
Cranmer, R. R.	Minneapolis
Cranston, R. W.	St. Louis Park
Curtin, J. F.	Minneapolis
Cutts, George	Minneapolis
Dady, E. E.	Minneapolis
Dahl, E. O.	Minneapolis
Dahl, J. A.	Minneapolis
Daniel, D. H.	Minneapolis
Daniel, L. M.	Minneapolis
Dart, L. O.	Minneapolis
Devereaux, T. J.	Wayzata
*Deziel, Godfrey	Minneapolis
Diehl, H. S.	Minneapolis
Diessner, H. D.	Minneapolis
Donaldson, C. A.	Mesa, Arizona
Dorge, R. I.	Minneapolis
Dornblaser, H. B.	Minneapolis
Dorsey, G. C.	Minneapolis
Doxey, G. L.	Minneapolis
Doyle, L. O.	Minneapolis
Drake, C. R.	Minneapolis
Dreisbach, Norman	Minneapolis
Drill, H. E.	Hopkins
Duff, E. R.	Minneapolis
Dunlap, E. H.	Minneapolis
Dunn, G. R.	Minneapolis
Duryea, Marry	Minneapolis
Dutton, C. E.	Minneapolis
Dwan, P. F.	Minneapolis
Dworsky, S. D.	Minneapolis
Ehrenberg, C. J.	Minneapolis
Ehrlich, S. P.	Minneapolis
Eich, Matthew	Minneapolis
Eisenstadt, D. H.	Minneapolis
Etel, G. D.	Minneapolis
Ellison, D. E.	Minneapolis

Emond, A. J.	Minneapolis
Erb, F. A.	Minneapolis
Erdman, C. A.	Minneapolis
Erickson, R. F.	Minneapolis
Ericson, J. G.	Minneapolis
Ericson, R. M.	Minneapolis
Evaas, E. T.	Minneapolis
Evans, R. D.	Minneapolis
Exley, E. W. F.	Minneapolis
Fahr, G. E.	Minneapolis
Fansier, W. A.	Minneapolis
Feehey, J. M.	Minneapolis
Fenger, E. P. K.	Oak Terrace
Fetterly, Warren	Minneapolis
Fink, L. W.	Minneapolis
Fink, W. H.	Minneapolis
Fitzgerald, D. F.	Minneapolis
Fjeldstad, C. A.	Minneapolis
Fowler, L. H.	Minneapolis
Fredericks, G. M.	Minneapolis
Friedell, Aaron	Minneapolis
Funk, V. K.	Oak Terrace
Ford, W. H.	Minneapolis
Gammell, J. H.	Minneapolis
Gardner, E. L.	Minneapolis
Geist, E. S.	Minneapolis
Giere, E. O.	Minneapolis
Giere, J. C.	Minneapolis
Giere, R. W.	Minneapolis
Giessler, P. W.	Minneapolis
Gilles, F. L.	Minneapolis
Ginsberg, Harry	Minneapolis
Gratzek, F. R.	Minneapolis
Grave, Floyd	Minneapolis
Green, E. K.	Minneapolis
Greene, W. P.	Minneapolis
Grimes, Marian	Minneapolis
Gunderson, N. A.	Minneapolis
Gustafson, H. T.	Minneapolis
Hacking, F. H.	Minneapolis
Haddow, N. W.	Minneapolis
Hagen, G. L.	Minneapolis
Haggard, G. D.	Minneapolis
Hall, J. M.	Minneapolis
Hallberg, C. A.	Minneapolis
Hamel, A. L.	Minneapolis
Hamilton, A. S.	Minneapolis
Hamlin, G. B.	Minneapolis
Hammond, A. J.	Minneapolis
Hannah, H. B.	Minneapolis
Hansen, C. O.	Minneapolis
Hansen, E. W.	Minneapolis
Hansen, Olga S.	Minneapolis
Hanson, H. J.	Minneapolis
Hanson, H. V.	Minneapolis
Hanson, W. A.	Minneapolis
Hare, E. R.	Minneapolis
Harrington, C. D.	Minneapolis
Harrington, F. E.	Minneapolis
Hartzell, T. B.	Minneapolis
Hastings, D. R.	Minneapolis
Haverfield, Addie R.	Minneapolis
Hawkinson, R. P.	Robbinsdale
Hayes, J. M.	Minneapolis
Head, D. P.	Minneapolis

\*Deceased.

## ROSTER OF MINNESOTA STATE MEDICAL ASSOCIATION

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Head, G. D.	Minneapolis	Macnie, J. S.	Minneapolis	Rochford, W. E.	Minneapolis
Hearn, W. O.	Minneapolis	Maland, C. O.	Minneapolis	Rodda, F. C.	Minneapolis
Hedback, A. E.	Minneapolis	Mann, A. T.	Minneapolis	Rodgers, C. L.	Minneapolis
Hem, R. R.	Minneapolis	Mariette, E. S.	Oak Terrace	Rosen, Samuel	Minneapolis
Hek, H. H.	Minneapolis	Mark, D. B.	Minneapolis	Rosenwald, R. M.	Minneapolis
Hendrickson, J. F.	Minneapolis	Matchan, G. R.	Minneapolis	Rucker, C. W.	Minneapolis
Henry, C. E.	Minneapolis	Matthews, Justus	Minneapolis	Rucker, W. H.	Minneapolis
Henry, M. O.	Minneapolis	Mattil, P. M.	Oak Terrace	Kud, N. E.	Minneapolis
Herman, A. L.	Minneapolis	Mattson, Hamlin	Minneapolis	Rudell, G. L.	Minneapolis
Hedorffer, M. B.	Minneapolis	Maxeiner, S. R.	Minneapolis	Russeth, A. N.	Minneapolis
Hiebert, J. P.	Minneapolis	May, W. H.	Minneapolis	Saliterman, B. I.	Minneapolis
Higgins, J. H.	Minneapolis	McCarthy, Donald	Minneapolis	Salt, C. G.	Minneapolis
Hill, Eleanor J.	Minneapolis	McDaniel, Orianna	Minneapolis	Sawatzky, W. A.	Minneapolis
Hirschfelder, A. D.	Minneapolis	McEachran, A.	Minneapolis	Schaaf, F. H. K.	Minneapolis
Hirschfield, F. R.	Minneapolis	McFarland, A. H.	Minneapolis	Schaefer, W. G.	Minneapolis
Hoagland, A. W.	Minneapolis	McGandy, R. F.	Minneapolis	Scheldrup, N. H.	Minneapolis
Hobbs, C. A.	Minneapolis	McGeary, G. E.	Minneapolis	Schmitt, A. F.	Minneapolis
Hodge, S. V.	Minneapolis	McInerney, Maurice	Minneapolis	Schmitt, S. C.	Minneapolis
Holand, A. S.	Minneapolis	McIntyre, George	Minneapolis	Schneider, J. P.	Minneapolis
Holl, P. M.	Minneapolis	McKinlay, C. A.	Minneapolis	Schussler, O. F.	Minneapolis
Holt, W. B.	Minneapolis	McKinley, J. C.	Minneapolis	Schwartz, V. J.	Minneapolis
Howard, W. H.	Minneapolis	McKinney, F. S.	Minneapolis	Schwyzier, Gustav	Minneapolis
Huetekens, E. J.	Minneapolis	McPheevers, H. O.	Minneapolis	Schwyzier, Robert	Minneapolis
Hughes, L. D.	Minneapolis	Meland, E. L.	Minneapolis	Scott, F. H.	Minneapolis
Hultkrans, R. E.	Minneapolis	Merkert, C. E.	Minneapolis	Seashore, Gilbert	Minneapolis
Hurd, Anna H.	Minneapolis	Merkert, G. L.	Minneapolis	Seham, Max	Minneapolis
Husband, M. W.	Minneapolis	Meyer, E. L.	Minneapolis	Sellestch, I. F.	Minneapolis
Hynes, J. E.	Minneapolis	Michael, J. C.	Minneapolis	Shapiro, M. J.	Minneapolis
Irwin, A. F.	Minneapolis	Michelson, H. E.	Minneapolis	Simons, Alma	Minneapolis
Jackson, C. M.	Minneapolis	Milton, J. S.	Minneapolis	Simpson, E. D.	Minneapolis
Jennings, F. L.	Oak Terrace	Moen, J. K., Jr.	Minneapolis	Siperstein, D. M.	Minneapolis
Jennings, Mary H.	Minneapolis	Moir, W. W.	Minneapolis	Sitar, R. F.	Minneapolis
Jensen, Harry	Minneapolis	Moorhead, M. B.	Minneapolis	Sivertsen, Ivar	Minneapolis
Jensen, M. J.	Minneapolis	Moriarty, Cecile R.	Minneapolis	Slocumb, Maude S.	Minneapolis
Johnson, A. B.	Minneapolis	Morrison, A. W.	Minneapolis	Smith, A. E.	Minneapolis
Johnson, A. E.	Minneapolis	Morse, R. W.	Minneapolis	Smith, A. M.	Minneapolis
Johnson, J. A.	Minneapolis	Morton, H. McI.	Minneapolis	Smith, H. R.	Minneapolis
Johnson, Julius	Minneapolis	Murphy, I. J.	Minneapolis	Smith, N. M.	Minneapolis
Johnson, N. A.	Minneapolis	Murphy, Leo	Minneapolis	Smith, Wyman	Minneapolis
Johnson, Norman	Minneapolis	Myers, J. A.	Minneapolis	Soderlind, R. T.	Minneapolis
Johnson, R. A.	Minneapolis	Nathanson, M. H.	Minneapolis	Solhaug, S. B.	Minneapolis
Johnson, S. M.	Minneapolis	Nelson, H. F.	Minneapolis	Spano, J. P.	Minneapolis
Johnson, Y. T.	Minneapolis	Nelson, C. P.	Owatonna	Spratt, C. N.	Minneapolis
Jones, G. M.	Minneapolis	Nelson, Harvey	Minneapolis	Stelter, L. A.	Minneapolis
Jones, H. W.	Minneapolis	Nelson, H. S.	Minneapolis	Stewart, C. A.	Minneapolis
Jones, W. K.	Minneapolis	Nelson, O. E.	Minneapolis	Stewart, R. L.	Minneapolis
Josewicz, Alexander	Minneapolis	Newhart, Horace	Minneapolis	Stomel, Joseph	Minneapolis
Kelby, G. M.	Minneapolis	Nordin, G. T.	Minneapolis	Strachauer, A. C.	Minneapolis
Kennedy, C. C.	Minneapolis	Nordland, Martin	Minneapolis	Strout, E. S.	Minneapolis
Kennedy, Jane F.	Minneapolis	Noth, H. V.	Minneapolis	Sturre, J. R.	Minneapolis
Kennedy, R. R.	Minneapolis	Nystrom, Ruth	Minneapolis	Sundt, Mathias	Minneapolis
Kertesz, G.	Minneapolis	Oberg, C. M.	Minneapolis	Swanson, Cephas	Minneapolis
Kibbe, O. A.	Minneapolis	O'Brien, W. A.	Minneapolis	Swanson, R. E.	Minneapolis
King, E. A.	Minneapolis	O'Donnell, J. E.	Minneapolis	Sweetser, H. B., Sr.	Minneapolis
King, H. T.	Minneapolis	Olson, F. A.	Minneapolis	Sweetser, H. B., Jr.	Minneapolis
King, W. R.	Minneapolis	Olson, O. A.	Minneapolis	Sweetser, T. H.	Minneapolis
Kinsella, T. J.	Oak Terrace	Olson, R. G.	Minneapolis	Sweitzer, S. E.	Minneapolis
Kistler, A. J.	Minneapolis	Oppen, E. G.	Minneapolis	Swendseen, C. G.	Minneapolis
Kistler, C. M.	Minneapolis	Owre, Oscar	Minneapolis	Taft, J. O.	Minneapolis
Knight, R. R.	Minneapolis	Parks, A. H.	Minneapolis	Tanner, A. C.	Minneapolis
Knight, R. T.	Minneapolis	Patterson, W. E.	Minneapolis	Ternstrom, O. H.	Minneapolis
Koch, J. C.	Minneapolis	Pauslen, E. L.	Minneapolis	Thomas, G. E.	Minneapolis
Koeckpe, G. M.	Minneapolis	Pearce, N. O.	Minneapolis	Thomas, G. H.	Minneapolis
Koller, H. M.	Minneapolis	Pederson, Harold	Minneapolis	Thomas, G. J.	Minneapolis
Koller, L. R.	Minneapolis	Pederson, R. M.	Minneapolis	Tingdale, A. C.	Minneapolis
Kucera, F. J.	Hopkins	Peppard, T. A.	Minneapolis	Truemann, H. S.	Minneapolis
Kucera, W. J.	Minneapolis	Perry, R. St. J.	Minneapolis	Tunstead, H. J.	Minneapolis
Lageresen, R. W.	Minneapolis	Petersen, J. K.	Minneapolis	Turnacliff, D. D.	Minneapolis
Lajoinie, J. M.	Minneapolis	Petersen, Thorvald	Minneapolis	Tyrrell, C. C.	Minneapolis
Lapierre, A. P.	Minneapolis	Peterson, H. W.	Minneapolis	Ude, W. H.	Minneapolis
Lapierre, C. A.	Minneapolis	Peterson, O. H.	Minneapolis	Ulrich, H. L.	Minneapolis
Larson, C. M.	Minneapolis	Peterson, W. C.	Minneapolis	Urner, J. A.	Minneapolis
Larson, L. M.	Minneapolis	Pettit, L. J.	Minneapolis	Vik, A. E.	Minneapolis
Laurent, A. A.	Minneapolis	Pettit, C. W.	Minneapolis	Voyer, E. O.	Minneapolis
LeVake, R. T.	Minneapolis	Pfunder, M. C.	Minneapolis	Wahlquist, H. F.	Minneapolis
Lazar, H. L.	Minneapolis	Phelps, K. A.	Minneapolis	Waldron, C. W.	Minneapolis
Lebowks, J. A.	Minneapolis	Platou, E. S.	Minneapolis	Wall, C. R.	Minneapolis
Lee, H. M.	Minneapolis	Pollard, D. W.	Minneapolis	Wangensteen, O. H.	Minneapolis
Leland, H. R.	Minneapolis	Pollock, D. K.	Minneapolis	Ward, A. W.	Minneapolis
Leland, M. N.	Minneapolis	Poppe, F. H.	Minneapolis	Ward, P. A.	Minneapolis
Leonard, L. J.	Minneapolis	Pratt, F. J.	Minneapolis	Warkam, T. T.	Minneapolis
Lillehei, E. J.	Robbinsdale	Pratt, J. A.	Minneapolis	Watson, J. A.	Minneapolis
Lind, C. J.	Minneapolis	Preine, I. A.	Minneapolis	Webb, R. C.	Minneapolis
Lindquist, R. H.	Minneapolis	Prim, J. A.	Minneapolis	Weisman, S. A.	Minneapolis
Linner, H. P.	Minneapolis	Proshok, C. E.	Minneapolis	Welles, H. J.	Minneapolis
Linton, W. B.	Minneapolis	Polzak, J. A.	Minneapolis	Westman, R. T.	Minneapolis
Litman, A. B.	Minneapolis	Quinby, T. F.	Minneapolis	Wethall, A. G.	Minneapolis
Litzenberg, J. C.	Minneapolis	Quist, H. W.	Minneapolis	Wetherby, Macnider	Minneapolis
Logefeld, R. C.	Minneapolis	Regnier, E. A.	Winneapolis	Weum, T. W.	Minneapolis
Long, Jesse	Minneapolis	Reynolds, J. S.	Minneapolis	White, S. M.	Minneapolis
Louis, E. A.	Minneapolis	Rice, C. O.	Minneapolis	White, W. D.	Minneapolis
Lundgreen, A. C.	Minneapolis	Richdorf, L. F.	Minneapolis	Widen, W. F.	Minneapolis
Lundquist, E. F.	Minneapolis	Ridgway, Florence	Minneapolis	Wiese, H. F. B.	Minneapolis
Lynch, M. J.	Minneapolis	Rigler, L. G.	Minneapolis	Wilcox, A. E.	Minneapolis
Lyon, E. P.	Minneapolis	Rishmiller, J. H.	Minneapolis	Wilder, R. L.	Minneapolis
Lysne, Henry	Minneapolis	Rizer, R. I.	Minneapolis	Wilken, P. A.	Minneapolis
MacDonald, A. E.	Minneapolis	Roan, C. M.	Minneapolis	Wilkerson, V. A.	Minneapolis
MacDonald, D. A.	Minneapolis	Robb, E. F.	Minneapolis	Willcutt, C. E.	Minneapolis
MacDonald, I. C.	Minneapolis	Roberts, T. S.	Minneapolis	Williams, H. L., Jr.	Minneapolis
Mach, F. B.	Minneapolis	Roberts, W. B.	Minneapolis	Williams, Robert	Minneapolis
		Robitshek, E. C.	Minneapolis	Winther, Nora M. C.	Minneapolis

[May, 1933]

Witham, C. A.	Minneapolis
Wittich, F. W.	Minneapolis
Wohlrabe, A. A.	Minneapolis
Wood, D. F.	Minneapolis
Woodworth, Elizabeth	Minneapolis

Wright, C. B.	Minneapolis
Wright, C. D.	Minneapolis
Wright, F. R.	Minneapolis
Wyatt, O. S.	Minneapolis
Wynne, H. M. N.	Minneapolis

Ylvisaker, R. S.	Minneapolis
Yoerg, O. W.	Minneapolis
Zaworski, E. A.	Minneapolis
Zierold, A. A.	Minneapolis
Ziskin, Thomas	Minneapolis

## KANDIYOHI-SWIFT COUNTY MEDICAL SOCIETY

Regular meetings, monthly at call  
Annual meeting, December

President	
Jensen, Herman H.	Atwater
Secretary	Benson
Scofield, C. L.	Benson
Anderson, R. E.	Willmar
Arnson, J. M.	Willmar
Behmier, F. W.	Appleton
Branton, A. F.	Willmar

Branton, B. J.	Willmar
Daignault, Oscar	Benson
Dowsell, W. J.	Kerkhoven
Fiksdal, M. J.	Willmar
Fredrickson, Alice C.	Lake Lillian
Fredrickson, G. U. Y.	Lake Lillian
Frisch, F. P.	Willmar
Frost, E. H.	Willmar
Giere, S. W.	Benson

Hodapp, R. J.	Willmar
Hutchinson, Henry	New London
Jacobs, J. C.	Willmar
Jensen, H. H.	Atwater
Johnson, Hans	Kerkhoven
Kaufman, W. C.	Appleton
Scofield, C. L.	Benson
Smith, B. F.	Willmar
Thompson, Arthur	Raymond

## LYON-LINCOLN COUNTY MEDICAL SOCIETY

Regular meetings, first Tuesday of month  
Annual meeting, October

President	Hendricks
Olson, Archibald	Hendricks
Secretary	
Workman, H. M.	Tracy
Akester, Ward	Marshall
Bossingham, O. N.	Lake Benton
Ford, B. C.	Marshall

Germo, Charles	Balaton
Golden, C. M.	Tyler
Gray, F. D.	Marshall
Happe, L. J.	Marshall
Hermanson, P. E.	Hendricks
Hoidal, A. D.	Tracy
Jacquot, G. L.	Marshall
Olson, Archibald	Hendricks
Persons, C. E.	Marshall

Purvis, G. H.	Russell
Robertson, J. B.	Cottonwood
Sanderson, E. T.	Minneota
Thordarson, Theo	Minneota
Vadheim, A. L.	Tyler
Valentine, W. H.	Tracy
Workman, H. M.	Tracy
Workman, W. G.	Tracy
Yaeger, W. W.	Ivanhoe

## MCLEOD COUNTY MEDICAL SOCIETY

Annual meeting, January

President	Hutchinson
Scholpp, O. W.	Hutchinson
Secretary	
Klima, W. W.	Stewart

Crow, E. R.	Arlington
Holm, H. H.	Glencoe
Jensen, A. H.	Hutchinson
Klima, W. W.	Stewart
Langhoff, A. H.	Glencoe
Lipmann, E. W.	Hutchinson
McMahon, M. J.	Green Isle

Ninneman, N. N.	Silver Lake
Sahr, W. G.	Hutchinson
Scholpp, O. W.	Hutchinson
Sheppard, Fred	Hutchinson
Sheppard, P. E.	Hutchinson
Trutna, T. J.	Silver Lake

## MEEKER COUNTY MEDICAL SOCIETY

Annual meeting, December

President	Dassel
Dulude, S. S.	Dassel
Secretary	
Danielson, K. A.	Litchfield

Brigham, Frank	Watkins
Danielson, K. A.	Litchfield
Danielson, Lennox	Litchfield
Dulude, S. S.	Dassel
Edwards, G. C.	Grover City

Macklin, W. E., Jr.	Litchfield
O'Connor, D. C.	Eden Valley
Telford, V. J.	Litchfield
Wilmot, H. E.	Litchfield

## MOWER COUNTY MEDICAL SOCIETY

Regular meetings, last Thursday of month except June, July and August  
Annual meeting, last Thursday of November

President	Austin
Havens, J. G. W.	Austin
Secretary	
Robertson, P. A.	Austin
Allen, A. W.	Austin
Allen, C. C.	Austin
Coleman, F. B.	Austin

Cronwell, B. J.	Austin
Flanagan, L. G.	Austin
Greene, H. H.	Austin
Grise, W. B.	Austin
Havens, J. G. W.	Austin
Hegge, O. H.	Austin
Hegge, R. S.	Austin
Henslin, A. E.	Le Roy
Hertel, G. E.	Austin

Leck, P. C.	Austin
Lommen, P. A.	Austin
McKenna, J. K.	Austin
Melzer, G. R.	Lyle
Mitchell, R. S.	Grand Meadow
Morse, M. F.	Le Roy
Rebman, E. C.	Austin
Robertson, P. A.	Austin
Sherman, H. T.	Grand Meadow

## NICOLLET-LE SUEUR COUNTY MEDICAL SOCIETY

Annual meeting, December

President	St. Peter
Lenander, M. E.	St. Peter
Secretary	
Petersen, M. C.	St. Peter
Aitkins, H. B.	Le Center

Covell, W. W.	St. Peter
Daniels, J. W.	St. Peter
Ericson, Swau	Le Sueur
Freeman, G. H.	St. Peter
Gully, R. J.	St. Peter
Holtan, Theodore	Waterville
Kerschbaumer, Louisa	St. Peter

Lenander, M. E.	St. Peter
McKeon, J. O.	Montgomery
Petersen, M. C.	St. Peter
Strathern, F. P.	St. Peter
Watz, C. E.	Le Sueur
Wolner, O. H.	St. Peter

## ROSTER OF MINNESOTA STATE MEDICAL ASSOCIATION

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## OLMSTED-HOUSTON-FILLMORE COUNTY MEDICAL SOCIETY

Regular meetings, first Tuesday each odd month  
 Annual meeting, November

<b>President</b>	Eusterman, G. B.	Rochester	Fulcher, O. H.	Rochester	Meade, J. R.	Rochester
<b>Secretary</b>	Piper, M. C.	Rochester	Gaarde, F. W.	Rochester	Meyerding, H. W.	Rochester
Adams, R. T.	Mantorville	Rochester	Garvin, R. O.	Rochester	Millet, R. F.	Rochester
Adsen, A. W.	Kasson	Rochester	Ghormley, R. K.	Rochester	Mills, S. D.	Rochester
Affeldt, D. E.	Rochester		Gibson, G. G.	Rochester	Moersch, F. P.	Rochester
Albers, E. C.	Rochester		Giffin, H. Z.	Rochester	Moersch, H. J.	Rochester
Alderson, L. R.	Rochester		Gillespie, J. B.	Rochester	Mohardt, J. H.	Rochester
Allen, E. V.	Rochester		Gilpin, S. F., Jr.	Rochester	Montgomery, Hamilton	Rochester
Allen, R. B.	Rochester		Goodwin, T. W.	Rochester	Montgomery, Jimmie Ethel	Rochester
Allen, W. A.	Rochester		Gray, H. K.	Rochester	Morey, J. B.	Rochester
Alvarez, W. C.	Rochester		Grimes, A. E., Jr.	Rochester	Mueller, S. C.	Rochester
Amberg, Samuel	Rochester		Grinnell, W. B.	Preston	Murray, S. E.	Rochester
Anderson, C. M.	Rochester		Habein, H. C.	Rochester	Mussey, R. D.	Rochester
Anderson, C. R.	Rochester		Haines, S. F.	Rochester	Nass, H. A.	Mabel
Anderson, M. J.	Rochester		Hale, D. E.	Rochester	Nehring, J. P.	Preston
Anderson, N. E.	Harmony		Hallenbeck, D. F.	Rochester	New, G. B.	Rochester
Atkinson, Walter	Rochester		Hardgrave, M. A. F.	Rochester	Newell, C. E.	Rochester
Baker, H. R.	Hayfield		Hargrave, R. L.	Rochester	Ohlinger, L. B.	Chicago, Ill.
Balfour, D. C.	Rochester		Harper, F. R.	Rochester	O'Leary, P. A.	Rochester
Bannick, E. G.	Rochester		Harrington, S. W.	Rochester	Olson, E. A.	Pine Island
Barber, K. W.	Rochester		Hartman, H. R.	Rochester	Olson, P. F.	Rochester
Bargen, J. A.	Rochester		Havens, F. Z.	Rochester	Ongard, L. K., Sr.	Houston
Barker, N. W.	Rochester		Hebert, W. H. J.	Rochester	Ongard, L. K., Jr.	Houston
Barnes, A. R.	Rochester		Heck, F. J.	Rochester	Overton, L. M.	Rochester
Beiswanger, R. H.	Wykoff		Heiland, G. M.	Spring Grove	Pace, J. McL.	Rochester
Belote, G. B.	Caledonia		Heiland, J. W.	Spring Grove	Parker, H. L.	Rochester
Benedict, W. L.	Rochester		Helmholz, H. F.	Rochester	Parkhill, Edith M.	Rochester
Bennett, R. J., Jr.	Rochester		Hempstead, B. E.	Rochester	Pattee, G. L.	Rochester
Berger, E. H.	Rochester		Hench, P. S.	Rochester	Pemberton, J. de J.	Rochester
Berkman, D. M.	Rochester		Henderson, M. S.	Rochester	Peterson, V. L.	Rochester
Berkman, J. M.	Rochester		Hewitt, Edith S.	Rochester	Phillips, J. R.	Rochester
Bigelow, C. E.	Dodge Center		Heyerdale, O. C.	Rochester	Piper, M. C.	Rochester
Binger, M. W.	Rochester		Hines, E. A., Jr.	Rochester	Plummer, H. S.	Rochester
Boothby, W. M.	Rochester		Hoerner, M. T.	Rochester	Plummer, W. A.	Rochester
Boudry, M. O.	Rochester		Horton, B. T.	Rochester	Pollack, L. W.	Rochester
Bowing, H. H.	Rochester		Howell, L. P.	Rochester	Popp, W. C.	Rochester
Braasch, W. F.	Rochester		Howell, W. L.	Rochester	Prangren, A. D.	Rochester
Brav, E. A.	Rochester		Hyde, T. L.	Rochester	Prickman, L. E.	Rochester
Broders, A. C.	Rochester		Iber, F. C.	Rochester	Priestley, J. T.	Rochester
Brown, A. E.	Rochester		Imes, P. R.	Rochester	Quade, R. H.	Rochester
Brown, G. E.	Rochester		Johnson, Ellsworth	Rochester	Raab, J. E.	Rochester
Brown, P. W.	Rochester		Johnson, R. B.	Laneboro	Randall, L. M.	Rochester
Brown, R. W.	Cambridge		Johnson, Spencer	Rochester	Rivers, A. B.	Rochester
Bruenner, Bertram	Rochester		Johnson, W. R.	Rochester	Rixford, E. L.	Rochester
Brunsting, L. A.	Rochester		Johnston, H. H.	Rochester	Robertson, H. E.	Rochester
Buie, L. A.	Rochester		Joyce, G. T.	Rochester	Robins, C. R., Jr.	Rochester
Bumpus, H. C.	Rochester		Judd, E. S.	Rochester	Robinson, L. W.	Rochester
Butsch, W. L.	Rochester		Kegaries, D. L.	Rochester	Rogin, J. R.	Rochester
Cabot, C. M.	Rochester		Keith, N. M.	Rochester	Rosenow, E. C.	Rochester
Cabot, Hugh	Rochester		Kennedy, F. S.	Rochester	Rudolph, E. A.	Rochester
Cain, E. F.	Rochester		Kennedy, R. L. J.	Rochester	Rynearson, E. H.	Rochester
Caldwell, J. M., Jr.	Rochester		Keeler, E. D.	Rochester	Sanford, A. H.	Rochester
Camp, J. D.	Rochester		Kernohan, J. W.	Rochester	Scherer, R. G.	Rochester
Canfield, W. W.	Houston		Keyes, H. C.	Rochester	Sheldon, W. D.	Rochester
Castleton, K. B.	Rochester		Kilbourne, A. F.	Rochester	Slocumb, C. H.	Rochester
Church, G. T.	Rochester		Kirch, W. A. W.	Rochester	Smith, F. D.	Kasson
Cliley, E. I. L.	Rochester		Kirklin, B. R.	Rochester	Smith, F. L.	Rochester
Clark, A. L.	Rochester		Koelsche, G. A.	Rochester	Smith, H. L.	Rochester
Collins, D. C.	Rochester		Joyce, G. L.	Stewartville	Smith, N. D.	Rochester
Comfort, M. W.	Rochester		Lannin, J. C.	Mabel	Snell, A. M.	Rochester
Conner, H. M.	Rochester		Lansbury, John	Rochester	Spooner, A. D.	Rochester
Cook, F. N.	Rochester		Larson, P. N.	Rochester	Sprague, P. H.	Rochester
Cook, N. C.	Rochester		Leddy, E. T.	Rochester	Stacy, L. J.	Rochester
Costello, R. T.	Rochester		Lemon, W. S.	Rochester	Stafne, W. A.	Rochester
Counsellor, V. S.	Rochester		Lendrum, F. C.	Rochester	Stark, W. B.	Rochester
Craig, W. McK.	Rochester		Lester, G. L.	Rochester	Starkey, T. A.	Rochester
Crenshaw, J. L.	Minneapolis		Lillie, H. I.	Rochester	Stephenson, G. W.	Rochester
Crewe, J. E.	Rochester		Lillie, W. L.	Rochester	Steven, George	Byron
Curry, F. S.	Rochester		Lochead, D. C.	Rochester	Stevens, G. A. W.	Rochester
Darnall, C. M.	Rochester		Logan, A. H.	Rochester	Stuart, F. A., Jr.	Rochester
Davis, A. C.	Rochester		Loney, W. R. R.	Rochester	Stuck, W. G.	Rochester
Davis, I. G.	Rushford		Love, J. G.	Rochester	Stuhler, L. G.	Rochester
Desjardins, A. U.	Rochester		Luden, Georgine...Victoria, B. C., Can.	Rochester	Sutherland, C. G.	Rochester
Deuterman, J. L.	Rochester		Lundy, J. S.	Rochester	Sutton, L. F.	Mazepa
Dixon, C. F.	Rochester		Lymburner, R. M.	Rochester	Swart, H. A.	Rochester
Dolder, F. C.	Eyota		Macy, J. W.	Rochester	Thiessen, N. W.	Rochester
Drake, F. A.	Laishesboro		Magath, T. B.	Rochester	Thompson, G. J.	Rochester
Drenckhahn, C. H.	Rochester		Magee, H. R.	Rochester	Thorp, E. G.	Rochester
Drips, D. G.	Rochester		Malerich, J. A.	Caledonia	Tovell, R. M.	Rochester
Dunlap, H. F.	Passaic, N. J.		Mann, F. C.	Rochester	Vanzant, Frances R.	Rochester
Dunlop, J. G., Jr.	Rochester		Marble, W. P.	Rochester	Vinson, P. P.	Rochester
Ely, C. B.	Spring Valley		Massey, B. D.	Rochester	Voldeng, K. E.	Rochester
Edward, George	Canton		Masson, D. M.	Rochester	Voris, H. C.	Rochester
Emmett, J. L.	Rochester		Masson, J. C.	Rochester	Wagener, H. P.	Rochester
Eusterman, G. B.	Rochester		Mayo, C. H.	Rochester	Waldron, G. W.	Rochester
Evans, V. L.	Rochester		Mayo, C. W.	Rochester	Waller, L. M.	Rochester
Evarts, A. B.	Rochester		Mayo, J. G.	Rochester	Walters, Waltman	Rochester
Fawcett, C. E.	Stewartville		Mayum, C. K.	Rochester	Watkins, C. H.	Rochester
Figi, F. A.	Rochester		McCarty, W. C.	Rochester	Watson, J. R.	Rochester
Foor, C. G.	Rochester		McCoskey, C. F.	Rochester	Weber, H. M.	Rochester
Foster, R. F.	Rochester		McKaig, C. B.	Pine Island	Weir, J. F.	Rochester
Fricke, R. E.	Rochester		McQuiston, J. S.	Rochester	Wellbrook, W. L. A.	Rochester
			McRoberts, J. W.	Rochester	Welsh, A. L.	Rochester
					Wilbur, D. L.	Rochester
					Wilder, R. M.	Rochester

Wilkinsen, E. A. .... Rochester  
 Williams, R. V. .... Rushford  
 Willius, F. A. .... Rochester

Wilson, L. B. .... Rochester  
 Wolf, H. J. .... Rochester  
 Woltman, H. W. .... Rochester

Wood, G. T., Jr. .... Rochester  
 Wood, H. G. .... Rochester  
 Woodruff, C. W. .... Chatfield

#### PARK REGION DISTRICT AND COUNTY MEDICAL SOCIETY

Otter Tail, Wilkin, Grant and Douglas Counties

Regular meetings, second Wednesday of January, April, July and October

Annual meeting, second Wednesday in October

President -  
 Broker, W. S. .... Battle Lake  
  
 Secretary  
 Nelson, W. I. .... Underwood  
  
 Baker, A. C. .... Fergus Falls  
 Baker, N. H. .... Fergus Falls  
 Benepe, J. L. .... St. Paul  
 Boysen, Peter. .... Pelican Rapids  
 Broker, W. S. .... Battle Lake  
 Burnap, W. L. .... Fergus Falls

Combacker, L. C. .... Fergus Falls  
 Drought, W. W. .... Fergus Falls  
 Esser, John ..... Perham  
 Estrem, C. O. .... Fergus Falls  
 Elbow Lake  
 Handel, W. R. .... Alexandria  
 Heiberg, E. A. .... Fergus Falls  
 Howard, Laura K. .... Fergus Falls  
 Jacobs, G. C. .... Fergus Falls  
 Johnson, O. V. .... Fergus Falls  
 Kemp, M. W. .... Fergus Falls  
 Kierland, P. E. .... Alexandria  
 Lee, W. A. .... Fergus Falls

Leibold, H. H. .... Parkers Prairie  
 Lewis, A. J. .... Henning  
 Love, F. A. .... Carlos  
 Meckstroth, C. W. .... Brandon  
 Naegeli, Frank ..... Fergus Falls  
 Nelson, O. N. .... Battle Lake  
 Nelson, W. I. .... Underwood  
 Parson, L. R. .... Elbow Lake  
 Paulson, T. S. .... Fergus Falls  
 Satersmoen, Theo. .... Pelican Rapids  
 Sather, E. R. .... Alexandria  
 Serkland, J. C. .... Rothsay  
 Tanquist, E. J. .... Alexandria

#### RAMSEY COUNTY MEDICAL SOCIETY

Regular meetings, last Monday of every month except June, July and August

Annual meeting, last Monday in January

President  
 Oerting, Harry ..... St. Paul

Secretary  
 Schulze, A. G. .... St. Paul

Abbott, J. S. .... St. Paul  
 Ahrens, A. E. .... St. Paul  
 Ahrens, A. H. .... St. Paul  
 Alberts, M. W. .... St. Paul  
 Alden, J. F. .... St. Paul  
 Aldes, Harry ..... St. Paul  
 Alexander, F. H. .... St. Paul  
 Allen, Mason ..... St. Paul  
 Armstrong, J. M. .... St. Paul  
 Arquist, A. S. .... St. Paul  
 Aurelius, J. R. .... St. Paul  
 Backus, A. S. .... St. Paul  
 Bacon, D. K. .... St. Paul  
 Bacon, Knox ..... St. Paul  
 Bacon, L. C. .... St. Paul  
 Balcome, F. E. .... St. Paul  
 Barry, L. W. .... St. Paul  
 Barness, Nellie ..... St. Paul  
 Beadie, W. D. .... Cannon Falls  
 Beals, Hugh ..... St. Paul  
 Bell, C. C. .... St. Paul  
 Bennington, P. H. .... St. Paul  
 Bentley, N. P. .... St. Paul  
 Berrixford, P. D. .... St. Paul  
 Bickel, J. F. .... St. Paul  
 Binger, J. F. .... St. Paul  
 Birnberg, T. L. .... St. Paul  
 Bock, R. A. .... St. Paul  
 Beckmann, Egil ..... St. Paul  
 Bohland, E. H. .... St. Paul  
 Bole, R. S. .... St. Paul  
 Borg, J. F. .... St. Paul  
 Bouma, L. R. .... St. Paul  
 Brand, G. D. .... St. Paul  
 Bray, E. R. .... St. Paul  
 Briggs, J. F. .... St. Paul  
 Brodie, W. D. .... St. Paul  
 Brown, E. I. .... St. Paul  
 Brown, J. C. .... St. Paul  
 Burch, F. E. .... St. Paul  
 Burfend, G. H. .... St. Paul  
 Burns, F. W. .... St. Paul  
 Burns, R. M. .... St. Paul  
 Burton, C. G. .... St. Paul  
 Busher, H. .... St. Paul  
 Caldwell, J. P. .... St. Paul  
 Caldwell, K. S. .... St. Paul  
 \*Cameron, J. A. .... St. Paul  
 Campbell, J. E. .... South St. Paul  
 Carroll, W. C. .... St. Paul  
 Carter, F. G. .... St. Paul  
 Chatterton, C. C. .... St. Paul  
 Christiansen, A. .... St. Paul  
 Christison, J. T. .... St. Paul  
 Clark, T. C. .... Minneapolis  
 Colby, Woodard ..... St. Paul  
 Cole, W. H. .... St. Paul  
 Collie, H. G. .... St. Paul  
 Colvin, A. R. .... St. Paul  
 Connor, C. E. .... St. Paul  
 Countryman, R. S. .... St. Paul  
 Cowern, E. W. .... North St. Paul  
 Critchfield, L. R. .... St. Paul

Crump, J. W. .... St. Paul  
 Colligan, J. M. .... St. Paul  
 Darling, J. B. .... St. Paul  
 Daugherty, E. B. .... St. Paul  
 Daugherty, L. E. .... St. Paul  
 Davis, Herbert ..... St. Paul  
 Davis, William ..... St. Paul  
 Dedolph, Karl ..... St. Paul  
 Derauf, B. I. .... St. Paul  
 Dickson, T. H., Jr. .... St. Paul  
 Dittman, G. C. .... St. Paul  
 Donohue, P. F. .... St. Paul  
 Dovre, C. M. .... St. Paul  
 Drake, C. B. .... St. Paul  
 Dunn, J. N. .... St. Paul  
 Earl, G. A. .... St. Paul  
 Earl, R. O. .... St. Paul  
 Edlund, G. .... St. Paul  
 Ely, O. S. .... South St. Paul  
 Emerson, E. C. .... St. Paul  
 Endress, E. K. .... St. Paul  
 Engberg, E. J. .... St. Paul  
 Ernest, G. C. .... South St. Paul  
 Estelby, E. C. .... St. Paul  
 Fahey, E. W. .... St. Paul  
 Ferguson, J. C. .... St. Paul  
 Fesler, H. H. .... St. Paul  
 Flanagan, H. F. .... St. Paul  
 Fogarty, C. W. .... St. Paul  
 Fogelberg, E. J. .... St. Paul  
 Foley, F. E. B. .... St. Paul  
 Freeman, C. D. .... St. Paul  
 Gager, E. C. .... St. Paul  
 Garbrecht, Arthur ..... St. Paul  
 Gardiner, D. G. .... St. Paul  
 Gardner, W. P. .... St. Paul  
 Geer, E. K. .... St. Paul  
 Gehlen, J. N. .... St. Paul  
 Goist, G. A. .... St. Paul  
 Ghent, C. H. .... St. Paul  
 Ghent, M. M. .... St. Paul  
 Gibbs, E. C. .... St. Paul  
 Gilfillan, J. S. .... St. Paul  
 Ginsberg, William ..... St. Paul  
 Goltz, E. V. .... St. Paul  
 Grantz, H. W. .... St. Paul  
 Gratzek, Thomas ..... St. Paul  
 Gruenhagen, A. P. .... St. Paul  
 Hagaman, G. K. .... St. Paul  
 Hall, A. R. .... St. Paul  
 Hall, H. H. .... St. Paul  
 Hammes, E. M. .... St. Paul  
 Hammond, J. F. .... St. Paul  
 Harmon, G. E. .... St. Paul  
 Hartfiel, W. F. .... St. Paul  
 Hartley, E. C., Jr. .... St. Paul  
 Hauser, V. P. .... St. Paul  
 Hawkins, V. J. .... St. Paul  
 Heath, A. C. .... St. Paul  
 Heck, W. W. .... St. Paul  
 Hedenstrom, F. G. .... St. Paul  
 Hengstler, W. H. .... St. Paul  
 Hensel, C. N. .... St. Paul  
 Herrmann, E. T. .... St. Paul  
 Hesselgrave, S. S. .... St. Paul  
 Hilger, A. W. .... St. Paul  
 Hilger, D. D. .... St. Paul  
 Hilger, L. A. .... St. Paul  
 Hochfilzer, J. J. .... St. Paul

Hoff, Alfred ..... St. Paul  
 Hoffman, M. H. .... St. Paul  
 Holcomb, J. T. .... St. Paul  
 Holcomb, O. W. .... St. Paul  
 Holt, J. E. .... St. Paul  
 Howard, W. S. .... St. Paul  
 Hultkrans, J. C. .... St. Paul  
 Ide, A. W. .... St. Paul  
 Ikeda, Kano ..... St. Paul  
 Johnson, A. M. .... St. Paul  
 Johnson, H. C. .... St. Paul  
 Johnson, J. A. .... St. Paul  
 Johnson, R. G. .... St. Paul  
 Johnson, T. H. .... St. Paul  
 Jones, D. C. .... St. Paul  
 Jones, E. M. .... St. Paul  
 Kadesky, David ..... St. Paul  
 Kammon, G. R. .... St. Paul  
 Kannary, E. L. .... St. Paul  
 Kasper, E. M. .... St. Paul  
 Kelly, J. V. .... St. Paul  
 Kelly, P. H. .... St. Paul  
 Keneck, E. V. .... St. Paul  
 Kennedy, W. A. .... St. Paul  
 Kesting, Herman ..... St. Paul  
 King, G. L. .... St. Paul  
 King, Z. P. .... St. Paul  
 Klein, H. N. .... St. Paul  
 Knauff, M. K. .... St. Paul  
 Kvitrud, G. .... St. Paul  
 Langenderfer, F. V. .... St. Paul  
 Larsen, C. L. .... St. Paul  
 Lax, M. H. .... St. Paul  
 Leahy, Bartholomew ..... St. Paul  
 Leavenworth, R. O. .... St. Paul  
 Leitch, Archibald ..... St. Paul  
 Leonard, G. J. .... St. Paul  
 Lepak, J. A. .... St. Paul  
 Lerche, William ..... Cable, Wis.  
 Levin, Bert ..... St. Paul  
 Lewis, W. W. .... St. Paul  
 Lick, C. L. .... St. Paul  
 Lippman, H. S. .... St. Paul  
 Little, W. J. .... St. Paul  
 Lowe, E. R. .... South St. Paul  
 Lowe, T. A. .... South St. Paul  
 Lundholm, A. M. .... St. Paul  
 Madden, J. F. .... St. Paul  
 Martineau, J. L. .... St. Paul  
 Mattson, C. H. .... St. Paul  
 McBeath, E. C. .... New York, N. Y.  
 McCarthy, W. R. .... St. Paul  
 McCloud, C. N. .... St. Paul  
 McKeon, Owen ..... St. Paul  
 McLaren, Jennette M. .... St. Paul  
 McNevin, C. F. .... St. Paul  
 Meyerding, E. A. .... St. Paul  
 Moza, J. A. .... St. Paul  
 Mogilner, S. N. .... St. Paul  
 Molander, H. A. .... St. Paul  
 Moquin, Marie A. .... St. Paul  
 Moran, T. R. .... St. Paul  
 Morrissey, F. B. .... St. Paul  
 Mortenson, N. G. .... St. Paul  
 Moss, M. N. .... St. Paul  
 Moynihan, T. J. .... St. Paul  
 Muller, R. T. .... St. Paul  
 Myers, Thomas ..... St. Paul  
 Naegeli, A. E. .... St. Paul

\*Deceased.

## ROSTER OF MINNESOTA STATE MEDICAL ASSOCIATION

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chester	Neher, F. H.	St. Paul	Roy, Philemon	St. Paul	Stoeckmann, A. E.	St. Paul
Pennington	Nelson, L. A.	St. Paul	Ruhberg, G. N.	St. Paul	Stolpestad, H. L.	St. Paul
Carson	Nippert, H. T.	St. Paul	Rutherford, W. C.	St. Paul	Strate, G. E.	St. Paul
Landon	Noble, J. F.	St. Paul	Ryan, J. J.	St. Paul	Swanson, E. O.	St. Paul
Falls	Nordin, C. G.	St. Paul	Ryan, J. M.	St. Paul	Swanson, J. A.	St. Paul
Lake	Nye, Katherine A.	St. Paul	Ryan, M. E.	St. Paul	Swendson, J. J.	St. Paul
Wood	Nye, Lillian L.	St. Paul	Satterlund, V. L.	St. Paul	Tieberg, C. B.	St. Paul
Rapids	O'Connor, L. J.	St. Paul	Savage, F. J.	St. Paul	Tift, C. R.	St. Paul
Andria	Oerting, Harry	St. Paul	Schoch, R. B.	St. Paul	Tregigas, H. R.	South St. Paul
Orthsay	Odgen, Warner	St. Paul	Schons, Edward	St. Paul	Van Slyke, C. A.	St. Paul
Andria	Oilage, Justus	St. Paul	Schuldt, F. C.	St. Paul	Veirs, Dean	St. Paul
Prairie	Oilage, Justus, Jr.	St. Paul	Schulze, A. G.	St. Paul	Veirs, Ruby S.	St. Paul
Pennington	Olson, C. A.	St. Paul	Schwartz, Arnold	St. Paul	Von der Weyer, William	St. Paul
Carson	O'Reilly, B. E.	St. Paul	Scott, E. E.	St. Paul	Waas, C. W.	St. Paul
Landon	Ostergren, E. W.	St. Paul	Senkler, G. E.	St. Paul	Warnock, R. W.	St. Paul
Falls	Page, C. V.	St. Paul	Setzer, H. J.	St. Paul	Warren, E. L.	St. Paul
Lake	Pearson, F. R.	St. Paul	Shannon, W. R.	St. Paul	Webber, F. L.	St. Paul
Wood	Pedersen, A. H.	St. Paul	Shellman, J. L.	St. Paul	Welch, M. C.	St. Paul
Rapids	Perry, C. G.	St. Paul	Shillington, M. A.	St. Paul	Werner, O. S.	Cambridge
Andria	Peterson, V. N.	St. Paul	Short, Jacob	St. Paul	Wheeler, M. W.	St. Paul
Orthsay	Plondike, F. J.	St. Paul	Simon, B. F.	St. Paul	Waitacre, J. C.	St. Paul
Andria	Prendergast, H. J.	St. Paul	Singer, B. J.	St. Paul	Whitcomb, E. H.	St. Paul
Prairie	Ramsey, W. R.	St. Paul	Skinner, H. O.	St. Paul	Whitmore, Frank	St. Paul
Pennington	Richards, E. T. F.	St. Paul	Snyder, G. W.	St. Paul	Williams, C. K.	St. Paul
Carson	Richardson, H. E.	St. Paul	Sohlberg, O. L.	St. Paul	Williamson, G. A.	St. Paul
Landon	Ritchie, H. P.	St. Paul	Souter, B. B.	St. Paul	Wilson, J. A.	St. Paul
Falls	Rogers, J. T.	St. Paul	Sprafka, J. M.	St. Paul	Wilson, J. V.	St. Paul
Lake	Rosenberger, H. P.	St. Paul	Steinberg, C. L.	St. Paul	Winnick, J. B.	St. Paul
Wood	Rosenholz, Burton	St. Paul	Sternier, E. G.	St. Paul	Wold, K. C.	St. Paul
Rapids	Rosenthal, Robert	St. Paul	Stewart, Alexander	St. Paul	Youngren, E. R.	St. Paul
Andria	Rothrock, J. L.	St. Paul	Stierle, Adolph, Jr.	St. Paul	Zimmermann, H. B.	St. Paul
Orthsay	Rothschild, H. J.	St. Paul	Stinnette, S. E.	St. Paul		
Andria						

## RED RIVER VALLEY MEDICAL SOCIETY

Kittson, Marshall, Pennington, Polk, Red Lake, Roseau, Norman and Mahnomen Counties  
Regular meetings, second Tuesday in April, September and December

Annual meeting, second Tuesday in December

President	Hodgson, H. H.	Crookston	Delmore, J. L.	Roseau	Nelson, H. E.	Crookston
Secretary	Oppgaard, C. L.	Crookston	Dunlop, A. H.	Crookston	Norman, J. F.	Crookston
			Engstrand, O. J.	Minneapolis	Oppgaard, C. L.	Crookston
			Froats, C. W.	Halstad	Oppgaard, M. O.	Crookston
			Froehlich, H. W.	Minneapolis	Overend, K. V.	Hallock
			Henney, W. H.	McIntosh	Paradis, W. G.	Crookston
			Hodgson, H. H.	Crookston	Parsons, J. G.	Crookston
			Hollands, W. H.	Fisher	Reff, A. R.	Red Lake Falls
			Holmstrom, C. H.	Warren	Roy, J. A.	Fosston
			Holte, Halvor	Crookston	Shedlov, A.	Ada
			Kahala, Arthur	Pasadena, Calif.	Shelland, J. T.	Ada
			Kirk, G. P.	East Grand Forks	Smith, A. M.	Thief River Falls
			Leitch, N. M.	Varroard	Stuurmanns, S. H.	Erskine
			Locken, O. E.	Crookston	Swedenburg, A. W.	Thief River Falls
			Lynde, O. G.	Thief River Falls	Torgerson, W. B.	Oklee
			Mellby, O. F.	Thief River Falls	Turnbull, Robert	Fosston
			Mercil, W. F.	Crookston	Watson, N. M.	Red Lake Falls
			Morley, G. A.	Crookston	Wattam, G. S.	Warren
					Wilttrot, I. G.	Oslo

## REDWOOD-BROWN COUNTY MEDICAL SOCIETY

Regular meetings, first Tuesday of winter months  
Annual meeting, May

President	Seifert, O. J.	New Ulm	Hammermeister, T. F.	New Ulm	Rothenburg, J. C.	Springfield
Secretary	Meierding, W. A.	New Ulm	Jamieson, Earl	Walnut Grove	Saffert, C. A.	New Ulm
			Johnson, W. E.	Morgan	Schoch, J. L.	New Ulm
			Just, H. J.	Lafayette	Seifert, O. J.	New Ulm
			Kolset, C. D.	Sanborn	Shrader, J. S.	Wheaton
			Kusske, A. L.	New Ulm	Vogel, H. A. L.	New Ulm
			Lindahl, M. J.	Winthrop	Vogel, J. H.	New Ulm
			McLane, Evelyn G.	Sleepy Eye	Weiser, G. B.	New Ulm
			McLane, W. O.	Sleepy Eye	Wellcome, J. W. B.	Sleepy Eye
			Meierding, W. A.	New Ulm	Weichman, F. H.	Young America
			Peterson, R. A.	Vesta	Wohlrabe, Clarence	Springfield
			Reineke, G. F.	New Ulm	Wohlrabe, E. J.	Springfield

## RICE COUNTY MEDICAL SOCIETY

Annual meeting, December

President	Murdoch, J. M.	Faribault	Haessly, S. B.	Faribault	Robilliard, C. M.	Faribault
Secretary	Plonske, C. J.	Faribault	Hanson, A. M.	Faribault	Rohrer, C. A.	Waterville
			Haynes, A. L.	Faribault	Rudie, C. N.	Kenyon
			Huxley, F. R.	Faribault	Rumpf, C. W.	Faribault
			Kanne, C. W.	Faribault	Rumpf, W. H.	Faribault
			Kuske, A. W.	Faribault	Seeley, I. F.	Northfield
			Lane, Laura A.	Northfield	Smith, P. A.	Faribault
			Lende, Norman	Faribault	Stewart, Gwendolyn	Faribault
			Lexa, F. J.	Lonsdale	Stiles, Angie G.	Northfield
			Mayland, M. L.	Faribault	Thorson, O. P.	Northfield
			Meyer, P. F.	Faribault	Traeger, C. A.	Faribault
			Moses, Joseph, Jr.	Northfield	Warren, F. S.	Washington, D. C.
			Murdoch, J. M.	Faribault	Wilkowske, R. J.	Nerstrand
			Nickerson, W. S.	Faribault	Wilson, Warren	Northfield
			Plonske, C. J.	Faribault	Wilson, W. E.	Northfield

## ST. LOUIS COUNTY MEDICAL SOCIETY

St. Louis, Lake, Cook, Carlton and Itasca Counties  
 Regular meetings, second Thursday each month  
 Annual meeting, October

President	Duluth	Graham, David.....	Duluth	Monroe, P. B.....	Two Harbors
Scherer, C. A.	Duluth	Graham, R. D.....	Duluth	Mooney, L. P.....	Marble
Secretary	Duluth	Graham, Robert.....	Duluth	More, C. W.....	Eveleth
Fischer, M. McC.	Duluth	Graves, W. N.....	Duluth	Mors, C. R.....	Zumbrota
Adams, B. S.	Hibbing	Haney, C. L.....	Duluth	Nelson, E. H.....	Chisholm
Alexander, C. E.	Duluth	Hanover, R. D.....	Littlefork	Nicholson, M. A.....	Duluth
Armstrong, E. L.	Duluth	Harris, C. N.....	Hibbing	Olson, A. E.....	Duluth
Athens, G.	Duluth	Hatch, W. E.....	Duluth	Parker, O. W.....	Ely
Ayres, G. T.	Ely	Hathaway, S. J.....	Proctor	Pennie, D. F.....	Duluth
Bardon, Richard	Duluth	Hayes, M. F.....	Nashwauk	Peterson, E. N.....	Eveleth
Bartels, E. C.	Duluth	Heuberg, G. A.....	Nopeming	Power, J. E.....	Duluth
Berdez, G. L.	Duluth	Heimark, O. E.....	Duluth	Raadquist, C. S.....	Montevideo
Bergquist, K. E.	Duluth	Hilding, A. C.....	Duluth	Raiter, F. W. S.....	Cloquet
Bianco, A. J.	Duluth	Hirschboeck, F. J.....	Duluth	Raiter, R. F.....	Cloquet
Binet, H. E.	Grand Rapids	Husby, H. W.....	Floodwood	Robinson, J. M.....	Duluth
Blacklock, S. S.	Hibbing	Jacobson, Clarence.....	Chisholm	Rood, D. C.....	Duluth
Blakely, C. C.	Barnum	Jensen, T. J.....	Coleraine	Rouse, J. J.....	Nopeming
Boman, P. G.	Duluth	Jolin, F. B.....	Coleraine	Rowe, O. W.....	Duluth
Boyer, S. H.	Duluth	Keyes, C. R.....	Duluth	Rudie, P. S.....	Duluth
Braverman, N. J.	Duluth	Kiesling, I. H.....	Nashwauk	Ryan, W. J.....	Duluth
Bray, C. W.	Biwabik	Klein, Harry.....	Duluth	Samson, E. R.....	Chisholm
Bullen, F. W.	Hibbing	Kliman, F. E.....	Duluth	Sarf, O. E.....	Buhl
Burns, R. L.	Two Harbors	Knapp, F. N.....	Duluth	Schröder, C. H.....	Duluth
Cantwell, W. F.	International Falls	Kohlbry, C. O.....	Duluth	Seashore, D. E.....	Duluth
Carstens, C. F.	Hibbing	Kotchevar, F. R.....	Eveleth	Shapiro, E. Z.....	Duluth
Chapman, T. L.	Duluth	Kraft, Peter.....	Duluth	Shastid, T. H.....	Duluth
Cheney, E. L.	Duluth	Kuth, J. R.....	Duluth	Sinamarck, Andrew.....	Hibbing
Christensen, E. P.	Two Harbors	Laird, A. T.....	Nopeming	Slyfield, F. F.....	Duluth
Clement, Gage	Duluth	Lamont, J. G.....	Nopeming	Smith, C. M.....	Duluth
Collins, A. N.	Duluth	Leckband, Norbert.....	Buhl	Smith, E. K.....	Duluth
Collins, H. C.	Duluth	Lenont, C. B.....	Virginia	Smith, W. R.....	Grand Marais
Coventry, W. A.	Duluth	Lepak, F. J.....	Duluth	Spicer, F. W.....	Duluth
Davis, B. F.	Duluth	Litman, S. N.....	Duluth	Strather, M. L.....	Gilbert
Doolittle, L. E.	Duluth	Loofbourouw, E. H.....	Keewatin	Strobel, W. G.....	Duluth
Doyle, G. C.	Duluth	Lum, C. E.....	Duluth	Sutherland, H. N.....	Ely
Drenning, F. C.	Duluth	Macfarlane, P. H.....	Chisholm	Swenson, A. O.....	Duluth
Eckman, P. F.	Duluth	MacRae, G. C.....	Chisholm	Taylor, C. W.....	Duluth
Eklund, J. W.	Duluth	Magnay, F. H.....	Duluth	Tibbets, M. H.....	Duluth
Elias, F. J.	Duluth	Manley, J. R.....	Duluth	Tilderquist, D. L.....	Duluth
Emanuel, K. W.	Duluth	Martin, E. T.....	Duluth	Toohy, E. L.....	Duluth
Eppard, R. M.	Cloquet	Martin, W. C.....	Duluth	Urberg, S. E.....	Duluth
Fellows, M. F.	Duluth	Mayne, R. M.....	Duluth	Vercellini, C. E.....	Duluth
*Ferreira, G. J.	Duluth	McCarty, P. D.....	Ely	Walker, A. E.....	Duluth
Fischer, M. McC.	Duluth	McComb, C. F.....	Duluth	Webber, E. E.....	Duluth
Fischer, J. M.	Proctor	McCoy, Mary K.....	Duluth	West, E. J.....	Duluth
Forbes, R. S.	Duluth	McDonald, A. L.....	Duluth	Wheeler, D. W.....	Duluth
Gillespie, M. G.	Duluth	McHaffie, O. L.....	Duluth	Wilkinson, Stella.....	Duluth
Gillespie, N. H.	Duluth	McNutt, J. R.....	Duluth	Winter, J. A.....	Duluth
Goldish, D. R.	Duluth	Merriman, L. L.....	Duluth	Young, T. O.....	Duluth
Gowan, L. R.	Duluth	Miners, G. A.....	Deer River	Young, V. A.....	Duluth
		Moe, R. J.....	Duluth		

## SCOTT-CARVER COUNTY MEDICAL SOCIETY

Regular meetings, second Tuesday each month  
 Annual meeting, second Tuesday in June

President	Chaska	Halgren, H. A.....	Watertown	Ormond, Douglas.....	Waconia
Simons, B. H.	Chaska	Hebeisen, M. B.....	Chaska	Phillips, W. H.....	Jordan
Secretary	New Prague	Hospodarsky, L. J.....	New Prague	Reiter, H. W.....	Shakopee
Cervenka, C. F.	New Prague	Juergens, H. M.....	Belle Plaine	Schimelpfenig, G. T.....	Chaska
Buck, F. H.	Shakopee	Lightbourn, E. T.....	Jordan	Schneider, H. A.....	Jordan
Cervenka, C. F.	New Prague	McKeon, James.....	St. Paul	Simons, B. H.....	Chaska
Eklund, E. J.	Norwood	Maertz, W. F.....	New Prague	Simons, L. T.....	Shakopee
Emmerson, W. S.	Mayer	Martin, T. P.....	Arlington	Westerman, A. E.....	Montgomery
Fischer, H. P.	Shakopee	Nagel, H. D.....	Waconia	Westerman, F. C.....	Montgomery
Fischer, P. M.	Shakopee	Novak, E. E.....	New Prague	Woodworth, L. F.....	Le Center
		Olson, C. J.....	Belle Plaine	Wunder, H. E.....	Shakopee

## SOUTHWESTERN MINNESOTA MEDICAL SOCIETY

Pipestone, Rock, Murray, Nobles, Cottonwood and Jackson Counties  
 Regular meetings, May and October  
 Annual meeting, October

President	Lakefield	Dolan, C. P.....	Worthington	Portmann, W. C.....	Jackson
Rose, J. T.	Lakefield	Doms, H. C.....	Slayton	Rose, J. T.....	Lakefield
McKeown, E. G.	Pipestone	Hitchings, W. S.....	Lakefield	Schutze, E. S.....	Mountain Lake
Arnold, E. W.	Adrian	Kelling, L. F.....	Lakefield	Sherman, C. L.....	Luverne
Basinger, H. P.	Windom	Kendahl, A. M.....	Jasper	Slater, S. A.....	Worthington
Benjamin, W. G.	Mountain Lake	Kilbride, E. A.....	Worthington	Smallwood, J. T.....	Worthington
Bofenkamp, F. W.	Luverne	Larson, J. T.....	Lake Wilson	Sogge, L. L.....	Windom
Brown, A. H.	Pipestone	McCrea, J. M.....	Fulda	Stanley, C. R.....	Worthington
Chadbourne, A. G.	Heron Lake	McKeown, E. G.....	Pipestone	Thorson, E. O.....	Luverne
Cress, P. J.	Ellsworth	Mork, B. O.....	Worthington	Tofte, Josephine.....	Dawson
De Boer, Hermanus	Edgerton	Mork, B. O. Jr.....	Worthington	Waller, J. D.....	Wilmont
		Patterson, W. E.....	Westbrook	Williams, A. B.....	St. Paul
		Perti, A. L.....	Windom	Williams, L. A.....	Slayton
		Piper, W. A.....	Mountain Lake	Wright, C. O.....	Luverne

\*Deceased

## ROSTER OF MINNESOTA STATE MEDICAL ASSOCIATION

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## STEARNS-BENTON COUNTY MEDICAL SOCIETY

Regular meetings, third Thursday of the month  
 Annual meeting, third Thursday in December

<b>President</b>	Gelz, J. J.	St. Cloud	Gelz, J. J.	St. Cloud	Meyer, A. A.	Melrose
<b>Secretary</b>			Goehrs, H. W.	St. Cloud	Moynihan, A. F.	Sauk Center
Libert, J. N.		St. Cloud	Haberman, E.	Osakis	Rathbun, A. M.	Rice
Buening, J. B.	Albany		Hemstead, Werner	St. Cloud	Rathbun, C. A.	St. Cloud
Boardman, D. V.	St. Cloud		Kohler, D. W.	Clearwater	Richards, W. B.	St. Cloud
Brigham, C. F.	St. Cloud		Koop, S. H.	St. Joseph	Ridgway, Alexander	South Haven
Buscher, J. C.	St. Cloud		Kuhlmann, August	Melrose	Rydburg, W. C.	Brooten
Du Bois, J. A.	Sauk Center		Johnson, Walfrid	Sauk Center	Schatz, F. J.	St. Cloud
Du Bois, J. F.	Sauk Center		Jones, R. N.	St. Cloud	Sherwood, G. E.	Kimball
Engstrom, G. F.	Belgrade		Lewis, C. B.	St. Cloud	Stangl, Fred	St. Cloud
Freeman, W. L.	St. Cloud		Libert, J. N.	St. Cloud	Stickney, Charlotte A.	St. Cloud
Friesleben, William	Sauk Rapids		Mahowald, A.	Albany	Sutton, C. S.	St. Cloud
			McDowell, J. P.	St. Cloud	Wenner, W. T.	St. Cloud
					Zachman, A. H.	Melrose

## STEELE COUNTY MEDICAL SOCIETY

Regular meetings, second Tuesday odd months  
 Annual meeting, last meeting of year

<b>President</b>	McIntyre, J. A.	Owatonna	Farabaugh, C. L.	Owatonna	Nelson, E. J.	Owatonna
<b>Secretary</b>			Kreuzer, T. C.	Owatonna	Roberts, O. W.	Owatonna
Dewey, D. H.		Owatonna	McEnaney, C. T.	Owatonna	Schaefer, J. F.	Owatonna
Dewey, D. H.		Owatonna	McIntyre, J. A.	Owatonna	Senn, E. W.	Owatonna

## UPPER MISSISSIPPI MEDICAL SOCIETY

Aitkin, Crow Wing, Morrison, Cass, Todd, Wadena, Clearwater, Koochiching,  
 Hubbard and Beltrami Counties  
 Regular meetings, Spring, Summer, Fall  
 Annual meeting, January

<b>President</b>	Shannon, S. S.	Crosby	Grawn, F. A.	Northome	Marcum, E. H.	Bemidji
<b>Secretary</b>	Badeaux, G. I.	Brainerd	Groschupft, T. P.	Bemidji	Mark, Hilbert	Ah-Gwah-Ching
			Grose, F. N.	Clarissa	McHugh, R. F.	Aitkin
			Hanson, E. C.	Park Rapids	Miller, W. A.	New York Mills
			Hawkinson, L. F.	Brainerd	Mosby, M. E.	Browerville
			Healy, R. T.	Pierz	Moyer, R. E.	Bemidji
			Hendrickson, R. R.	Wadena	Nelson, Nesmith	Brainerd
			Holst, C. F.	Little Falls	Quanstrom, V. E.	Brainerd
			Holst, J. B.	Little Falls	Ringle, O. F.	Walker
			House, Z. E.	Cass Lake	Roberts, L. M.	Little Falls
			Hubbard, O. E.	Brainerd	Shannon, S. S.	Crosby
			Hubin, E. G.	Deerwood	Simons, E. J.	Swanville
			Jacobson, D. J.	Blackduck	Smith, B. A.	Crosby
			Johnson, E. W.	Bemidji	Smith, E. H.	Bemidji
			Kelly, B. W.	Aitkin	Stevens, John	Gonvik
			Kerlan, S. Z.	Aitkin	Thabes, J. A., Sr.	Brainerd
			Lamb, H. L.	Little Falls	Van Valkenburg, B. F.	Long Prairie
			Larson, L. M.	Oak Terrace	Watson, J. D.	Holdingford
			Laughlin, J. T.	Grey Eagle	Will, W. W.	Bertha
			Lund, W. J.	Staples	Withrow, M. E.	International Falls

## WABASHA COUNTY MEDICAL SOCIETY

Annual meeting, first Thursday after the first Monday in July

<b>President</b>	Stryker, W. B.	Plainview	Bayley, E. C.	Lake City	Frost, R. H.	Wabasha
<b>Secretary</b>			Bowers, H. E.	Lake City	Radabaugh, R. C.	Hastings
Wilson, W. F.		Lake City	Cochrane, W. J.	Lake City	Slocumb, J. A.	Plainview

## WASECA COUNTY MEDICAL SOCIETY

Regular meetings, at call of President  
 Annual meeting, third Friday in December

<b>President</b>	Gallagher, B. J.	Waseca	Bernstein, W. C.	New Richland	McIntire, H. M.	Waseca
<b>Secretary</b>			Gallagher, B. J.	Waseca	Oeljen, S. C. G.	Waseca
Tavenner, J. L.			Hagen, H. O.	New Richland	Swenson, O. J.	Waseca

Tavenner, J. L.		Janesville	St. Croix
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## WASHINGTON COUNTY MEDICAL SOCIETY

Regular meetings, second Tuesday of every month except June, July and August  
 Annual meeting, second Tuesday in December

<b>President</b>	Josewski, R. J.	Stillwater	Brekke, H. J.	Stillwater	Poirier, J. A.	Forest Lake
<b>Secretary</b>			Brooks, G. F.	Stillwater	Ruggles, G. McC.	Stillwater
Boley, E. S.		Stillwater	Haines, J. H.	Stillwater	Strand, E. V.	Bayport
Boley, E. S.		Stillwater	Humphrey, W. R.	Stillwater	Stuhl, J. W.	Stillwater

Kalinoff, D.	Stillwater	Marine-on-St. Croix
		Stillwater

Van Meier, Henry	Stillwater
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[May, 1933]

## WATONWAN COUNTY MEDICAL SOCIETY

Annual meeting, December

Bregel, F. L.	President	St. James	Bergman, O. B.	St. James	Hagen, O. E.	Butterfield
Secretary			Cainreude, E. J.	St. James	McCarthy, W. J.	Madelia
Grimes, H. B.		Madelia	Bregel, F. L.	St. James	Thompson, Albert	St. James

## WEST CENTRAL MINNESOTA MEDICAL SOCIETY

Big Stone, Traverse, Pope and Stevens Counties  
 Regular meetings, twelfth of January, April, July and October  
 Annual meeting, October 12

Giesen, A. F.	President	Starbuck	Bergan, Otto	Clinton	Giesen, A. F.	Starbuck
Secretary			Caine, C. E.	Morris	Karn, B. R.	Ortonville
Lindberg, A. L.		Wheaton	Eberlin, E. A.	Genwood	Lindberg, A. L.	Wheaton

## WINONA COUNTY MEDICAL SOCIETY

Regular meetings, first Monday in January, April, July and October  
 Annual meeting, first Monday in January

Keyes, J. D.	President	Winona	Dukelow, D. A.	St. Charles	Page, R. L.	St. Charles
Steiner, I. W.	Secretary	Winona	Heise, W. F. C.	Winona	Risser, E. D.	Winona
Benoit, F. T.		Winona	Keyes, E. D.	Winona	Robbins, C. P.	Winona
Christensen, E. E.		Winona	Keyes, J. D.	Winona	Satterlee, H. W.	Lewiston
Clay, F. H.		St. Charles	Lichtenstein, H.	Winona	Schaefer, S.	Winona
Dierkes, G. J.		Rollingstone	Mattison, P. A.	Winona	Steiner, I. W.	Winona
			McLaughlin, E. M.	Winona	Tweedy, G. J.	Winona
			Meinert, A. E.	Winona	Walker, G. H.	Winona
			Nauth, W. W.	Winona	Wilson, R. H.	Winona
			Neumann, C. A.	Winona	Younger, L. I.	Winona

## WRIGHT COUNTY MEDICAL SOCIETY

Regular meeting, first Tuesday after the first Monday quarterly  
 Annual meeting, October

Bendix, L. H.	President	Annandale	Bendix, L. H.	Annandale	Lee, J. L.	Watertown
Secretary			Catlin, J. J.	Buffalo	Olson, J. W.	Cokato
Catlin, J. J.		Buffalo	Ellison, F. E.	Monticello	Peterson, O. L.	Cokato
Anderson, W. P.		Buffalo	Harriman, L.	Howard Lake	Ridgway, A. M.	Annandale
			Hart, W. E.	Monticello	Roholt, C. L.	Waverly
			Johnson, V. P.	Delano	Rousseau, Victor	Maple Lake
			Klaveness, E.	St. Paul	Swezey, B. F.	Buffalo

## ALPHABETICAL ROSTER

Aanes, A. M.	Red Wing	Barr, W. H.	Wells	Bowers, J. T.	Thief River Falls
Abbott, J. S.	St. Paul	Barron, Moses	Minneapolis	Bowing, H. H.	Rochester
Ahorn, W. H.	Hawley	Barry, L. W.	St. Paul	Boyer, S. H.	Duluth
Abraham, A. L.	Gibbon	Barsness, Nellie	St. Paul	Boynton, Ruth	Minneapolis
Adams, B. S.	Hibbing	Bartels, E. C.	Duluth	Boysen, H.	Welcome
Adams, R. C.	Bird Island	Basinger, H. P.	Windom	Boysen, Peter	Pelican Rapids
Adams, R. T.	Mantorville	Bass, G. W.	Minneapolis	Braasch, W. F.	Rochester
Adkins, C. M.	Thief River Falls	Baxter, S. H.	Minneapolis	Bracken, H. M.	Claremont, Calif.
Adson, A. W.	Rochester	Bayley, E. C.	Lake City	Brand, G. D.	St. Paul
Afeldt, D. E.	Kasson	Beadie, W. D.	Cannon Falls	Brand, W. A.	Redwood Falls
Agnew, A. T.	International Falls	Beals, Hugh	St. Paul	Branham, D. S.	Albert Lea
Ahrens, A. E.	St. Paul	Beard, A. H.	Minneapolis	Branton, A. F.	Willmar
Ahrens, A. H.	St. Paul	Beard, R. O.	Minneapolis	Branton, B. J.	Willmar
Ahrens, R. S.	Minneapolis	Bedford, E. W.	Minneapolis	Bratrud, A. F.	Minneapolis
Aikens, H. B.	Le Center	Beede, E. R.	Fairbault	Bratrud, O. E.	Thief River Falls
Akester, Ward.	Marshall	Behmier, F. W.	Appleton	Bratrude, E. J.	St. James
Albers, E. C.	Rochester	Beise, R. A.	Brainerd	Brav, E. A.	Rochester
Alberts, M. W.	St. Paul	Beiswanger, R. H.	Wykoff	Braverman, N. J.	Duluth
Alden, J. F.	St. Paul	Bell, C. C.	St. Paul	Bray, C. W.	Biwabik
Alderson, L. R.	Rochester	Bell, E. T.	Minneapolis	Bray, E. R.	St. Paul
Aldes, Harry	St. Paul	Bell, J. W.	Minneapolis	Bregel, F. I.	St. James
Alexander, C. E.	Duluth	Belote, G. B.	Caledonia	Brekke, H. J.	Stillwater
Alexander, F. H.	St. Paul	Bendix, L. H.	Annandale	Briggs, J. F.	St. Paul
Alien, A. W.	Austin	Benedict, E. E.	Minneapolis	Brigham, C. F.	Watkins
Alien, C. C.	Austin	Benedict, W. L.	Rochester	Broders, A. C.	Rochester
Alien, E. V.	Rochester	Benepe, J. L.	St. Paul	Brodie, W. D.	St. Paul
Alien, H. W.	Minneapolis	Benham, E. W.	Mankato	Broker, W. S.	Battle Lake
Allen, Mason	St. Paul	Benjamin, A. E.	Minneapolis	Brooks, G. F.	Stillwater
Allen, R. B.	Rochester	Benjamin, W. G.	Pipestone	Brown, A. E.	Rochester
Allen, W. A.	Rochester	Benn, F. G.	Minneapolis	Brown, A. H.	Pipestone
Allison, R. G.	Minneapolis	Bennett, R. J., Jr.	Rochester	Brown, E. D.	Minneapolis
Almqvist, H. E.	Minneapolis	Bennion, F. H.	St. Paul	Brown, E. I.	St. Paul
Altnow, H. O.	Minneapolis	Benoit, F. T.	Winona	Brown, E. J.	Minneapolis
Alvarez, W. C.	Rochester	Benson, T. Q.	East Grand Forks	Brown, G. E.	Rochester
Amberg, Samuel.	Rochester	Bentley, N. P.	St. Paul	Brown, J. C.	St. Paul
Andersen, A. G.	Minneapolis	Berdez, G. L.	Duluth	Brown, L. L.	Crookston
Andersen, S. C.	Minneapolis	Bergan, Otto	Clinton	Brown, P. W.	Rochester
Anderson, C. M.	Rochester	Berge, D. O.	Roseau	Brown, R. W.	Cambridge
Anderson, C. R.	Rochester	Berger, E. H.	Rochester	Brownstone, Manuel	Sandstone
Anderson, D. D.	Minneapolis	Bergl, L. N.	Montevideo	Bruenner, Bertram	Rochester
Anderson, E. D.	Minneapolis	Bergheim, M. C.	Hawley	Brunsting, L. A.	Rochester
Anderson, E. R.	Minneapolis	Bergman, O. B.	St. James	Brusegard, J. F.	Red Wing
Anderson, F. J.	Minneapolis	Bergquist, K. E.	Duluth	Buck, F. H.	Shakopee
Anderson, J. K.	Minneapolis	Berkman, D. M.	Rochester	Buie, L. A.	Rochester
Anderson, K. W.	Minneapolis	Berkman, J. M.	Rochester	Bulkley, Kenneth	Minneapolis
Anderson, M. J.	Rochester	Berkowitz, N. J.	Minneapolis	Bullard, Mattie J.	Minneapolis
Anderson, N. E.	Harmony	Bernard, B. C.	Thief River Falls	Bullen, F. W.	Hibbing
Anderson, P. A.	Minneapolis	Bernstein, W. C.	New Richland	Bumpus, H. C.	Rochester
Anderson, R. E.	Willmar	Berrisdorf, P. D.	St. Paul	Burch, F. E.	St. Paul
Anderson, S. H.	Red Wing	Bertelson, O. L.	Crookston	Burfiend, G. H.	St. Paul
Anderson, U. S.	Minneapolis	Bessesen, A. N., Sr.	Minneapolis	Burnap, W. L.	Fergus Falls
Anderson, W. P.	Buffalo	Bessesen, A. N., Jr.	Minneapolis	Burns, F. W.	St. Paul
Anderson, W. S.	Minneapolis	Bessesen, D. H.	Minneapolis	Burns, H. D.	Albert Lea
Andreassen, E. C.	Minneapolis	Bessesen, W. A.	Minneapolis	Burns, M. A.	Milan
Andrews, R. N.	Mankato	Beuning, J. B.	Albany	Burns, R. L.	Two Harbors
Annis, H. B.	Minneapolis	Bianco, A. J.	Duluth	Burns, R. M.	St. Paul
Archibald, F. M.	Mahnomen	Bicek, J. F.	St. Paul	Burton, C. G.	St. Paul
Arends, A. L.	Askov	Bigelow, C. E.	Dodge Center	Buscher, J. C.	St. Cloud
Arey, H. C.	Excelsior	Billings, R. E.	Franklin	Busher, H.	St. Paul
Armstrong, E. L.	Duluth	Binet, H. E.	Grand Rapids	Butler, John.	Minneapolis
Armstrong, J. M.	St. Paul	Binger, H. E.	St. Paul	Butsch, W. L.	Rochester
Arnold, E. W.	Adrian	Binger, M. W.	Rochester	Butturff, C. R.	Freeborn
Arnquist, A. S.	St. Paul	Birnberg, T. L.	St. Paul	Butz, J. A.	Monterey
Arnson, J. M.	Benson	Black, William	Mankato	Butzer, J. A.	Mankato
Arvidson, C. G.	Minneapolis	Blacklock, S. S.	Hibbing	Buzzelle, L. K.	Minneapolis
Athens, A. G.	Duluth	Blake, James.	Hopkins	Cable, M. L.	Minneapolis
Atkinson, Walter.	Rochester	Blakely, C. C.	Barnum	Cabot, C. M.	Rochester
Aune, Martin	Minneapolis	Blanchard, H. G.	Fairmont	Cabot, G. S.	Minneapolis
Aurand, W. H.	Minneapolis	Blaustone, H. H.	Minneapolis	Cabot, Hugh	Rochester
Aurelius, J. R.	St. Paul	Blegen, H. M.	Warren	Cabot, V. S.	Minneapolis
Avery, J. F.	Minneapolis	Blumenthal, J. S.	Minneapolis	Cady, L. H.	Minneapolis
Ayers, G. T.	Ely	Boardman, D. V.	St. Cloud	Cain, E. F.	Rochester
Babcock, F. M.	Northfield	Bock, R. A.	St. Paul	Caine, C. E.	Morris
Backus, A. S.	St. Paul	Bockman, M. W. H.	Minneapolis	Caldwell, J. M., Jr.	Rochester
Bacon, D. K.	St. Paul	Boeckmann, Egil	St. Paul	Caldwell, J. P.	St. Paul
Bacon, Knox	St. Paul	Bofenkamp, F. W.	Luverne	Caldwell, K. S.	St. Paul
Bacon, L. C.	St. Paul	Bohl, G. W.	Ada	Calhoun, F. W.	Albert Lea
Bacon, R. S.	Montevideo	Bohland, E. H.	St. Paul	Callahan, F. F.	Pokegama
Badeaux, G. I.	Brainerd	Boies, L. R.	Minneapolis	Callstrom, G. W.	Minneapolis
Baley, H. B.	Fairmont	Bole, R. S.	St. Paul	Cameron, Isabell.	Minneapolis
Baker, M. P.	Minneapolis	Boleyn, E. S.	Stillwater	*Cameren, J. A.	St. Paul
Baker, A. C.	Fergus Falls	Boman, P. G.	Duluth	Camp, J. D.	Rochester
Baker, A. T.	Minneapolis	Booth, A. E.	Minneapolis	Camp, W. E.	Minneapolis
Baker, E. L.	Minneapolis	Boothby, W. M.	Rochester	Campbell, J. E.	South St. Paul
Baker, H. R.	Hayfield	Boreen, C. A.	Minneapolis	Campbell, L. M.	Minneapolis
Baker, Looe.	Minneapolis	Borg, J. F.	St. Paul	Campbell, O. J.	Minneapolis
Baker, N. H.	Fergus Falls	Borges, E. J.	Minneapolis	Campbell, R. A.	Minneapolis
Balcone, F. E.	St. Paul	Borgerson, A. H.	Hewitt	Canfield, W. W.	Houston
Balfour, D. C.	Rochester	Borreson, Baldwin	Ah-Gwah-Ching	Cantwell, W. F.	International Falls
Bannick, E. G.	Rochester	Bosland, H. G.	Verndale	Cardle, A. E.	Minneapolis
Barber, J. P.	Minneapolis	Bossingham, O. N.	Lake Benton	Carlaw, C. M.	Minneapolis
Barber, K. W.	Rochester	Bottolison, B. T.	Moorhead	Caron, R. P.	Minneapolis
Bardon, Richard.	Duluth	Boudry, M. O.	Rochester	Carroll, W. C.	St. Paul
Bargen, J. A.	Rochester	Bouma, L. R.	St. Paul	Carstens, C. F.	Hibbing
Barker, N. W.	Rochester	Bouman, H. A. H.	Minneapolis	Carter, F. G.	St. Paul
Barnes, A. R.	Rochester	Bowers, H. E.	Lake City	Castleton, K. B.	Rochester

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Catlin, J. J.	Buffalo	St. Paul	Perham
Cavanor, F. T.	Minneapolis	Roseau	Fergus Falls
Cervenka, C. F.	New Prague	Wells	Rochester
Chadbourne, A. G.	Heron Lake	Mankato	Ghormley
Chapman, T. L.	Duluth	St. Paul	Ghosts
Cheney, E. L.	Duluth	Rochester	Gibson
Cherry, C. H.	Minneapolis	St. Paul	Giere
Chesley, A. J.	Minneapolis	Rochester	Giere
Chatterton, C. C.	St. Paul	Wayzata	Giere
Christensen, E. P.	Two Harbors	Owatonna	Giere
Christensen, E. E.	Winona	St. Paul	Giesen
Christensen, C. R.	Minneapolis	Minneapolis	Giesler
Christiansen, A.	St. Paul	Rochester	Giffin
Christianson, H. W.	Minneapolis	St. Paul	Gilliland
Christison, J. T.	St. Paul	Rochester	Gilles
Church, G. T.	Rochester	Worthington	Gillespie
Cilley, E. I. L.	Rochester	Eyota	Gillespie
Clark, A. L.	Rochester	Slayton	Gilmor
Clark, H. S.	Minneapolis	Mesa, Ariz.	Gilpin
Clark, T. C.	Minneapolis	St. Paul	Ginsberg
Clay, F. H.	St. Charles	Duluth	Ginsberg
Claydon, D. R.	Red Wing	Sacred Heart	Gobirsch
Claydon, H. F.	Zumbrota	Minneapolis	Goehrs
Claydon, L. E.	Red Wing	Minneapolis	Golden
Clement, Gage	Duluth	Minneapolis	Goldish
Clement, J. B.	Lester Prairie	St. Paul	Goltz
Cochrane, W. J.	Lake City	Kerkhoven	Goodwin
Cohen, S. S.	Oak Terrace	Minneapolis	Gowan
Colby, Woodard	St. Paul	Duluth	Graham
Cole, H. B.	Redwood Falls	Minneapolis	Graham
Cole, W. H.	St. Paul	Minneapolis	Graham
Coleman, F. B.	Austin	Lanesboro	Graham
Colie, H. G.	St. Paul	Hinckley	Grant
Collins, A. N.	Duluth	Dredge, H. P.	Gratzek
Collins, D. C.	Rochester	Dreisbach, Norman	Gratzek
Collins, H. C.	Duluth	Drenckhahn, C. H.	Grave
Collins, J. S.	Wabasha	Drenning, F. C.	Graves
Colvin, A. R.	St. Paul	Droll, H. E.	Grawn
Combbacker, L. C.	Fergus Falls	Drought, W. W.	Gray, F.
Comfort, M. W.	Rochester	Dubbe, F. H.	Gray, F.
Condit, W. H.	Minneapolis	Du Bois, J. A.	Green, F.
Connor, H. M.	Rochester	Du Bois, J. F.	Greene
Connor, C. E.	St. Paul	Duclos, J. A.	Greene
Cook, E. N.	Rochester	Duff, E. R.	Greimes
Cook, H. W.	Minneapolis	Dugan, L. F.	Greimes
Cook, J. M.	Staples	Dukelow, D. A.	Grimmell
Cook, N. C.	Rochester	Dulude, S. S.	Grisse
Cooney, H. C.	Princeton	Dunay, N. S.	Groschupf
Cooperman, H. O.	Minneapolis	Dunlap, E. H.	Grose
Corbett, J. F.	Minneapolis	Dunlap, H. F.	Gruenbaum
Cornies, A. D.	Minneapolis	Dunlop, A. H.	Gullixson
Corrigan, J. E.	Spooner	Dunlop, J. G., Jr.	Gully, R.
Cosgriff, J. A.	Bird Island	Dunn, G. R.	Gundersen
Cosman, E. O.	Minneapolis	Dunn, J. N.	Gustafson
Costello, R. T.	Rochester	Durgin, F. L.	Haben
Cottam, G. G.	Minneapolis	Duryea, Marbry	Haberman
Counsellor, V. S.	Rochester	Dutton, C. E.	Hacking
Countryman, R. S.	St. Paul	Dwan, P. F.	Haddow
Cowell, W. W.	St. Peter	Dworsky, S. D.	Hagama
Coventry, W. A.	Duluth	Dysterheft, A. F.	Hagen
Cowern, E. W.	North St. Paul	Earl, G. A.	Hagen
Crafts, L. M.	Minneapolis	Earl, R. O.	Hagen
Craig, W. McK.	Rochester	Eberlin, E. A.	Hagen
Cranmer, R. R.	Minneapolis	Eby, C. B.	Hagen
Cranston, R. W.	St. Louis Park	Eckman, P. F.	Hagen
Cremer, M. H.	Red Wing	Eckstein, A. W.	Hagen
Cremer, P. H.	Hastings	Edlund, G.	Hagen
Crenshaw, J. L.	Rochester	Edward, George	Hagen
Cress, E. E.	Boyd	Edwards, G. C.	Hagen
Cress, P. J.	Ellsworth	Edwards, R. T.	Hagen
Crewe, J. E.	Rochester	Ehrenberg, C. J.	Hagen
Critchfield, L. R.	St. Paul	Eich, Matthew	Hagen
Cronwell, B. J.	Austin	Eisenstadt, D. H.	Hagen
Crow, E. R.	Arlington	Eitel, G. D.	Hagen
Crump, J. W.	St. Paul	Eikblad, J. W.	Hagen
Culligan, J. M.	St. Paul	Eiklund, E. J.	Hagen
Culver, L. G.	Thief River Falls	Elias, F. J.	Hagen
Curry, F. S.	Rochester	Ellison, D. E.	Hagen
Curtin, J. F.	Minneapolis	Ellison, F. E.	Hagen
Cutts, George	Minneapolis	Ely, O. S.	Hagen
Dady, E. E.	Minneapolis	Emanuel, K. W.	Hagen
Dahl, E. O.	Minneapolis	Emerson, E. C.	Hagen
Dahl, G. A.	Mankato	Emmerson, W. S.	Hagen
Dahl, J. A.	Minneapolis	Emmett, J. L.	Hagen
Daignault, Oscar	Benson	Emond, A. J.	Hagen
Daniel, D. H.	Minneapolis	Endress, E. K.	Hagen
Daniel, L. M.	Minneapolis	Engberg, E. J.	Hagen
Daniels, J. W.	St. Peter	Engstrand, O. J.	Hagen
Danielson, K. A.	Litchfield	Engstrom, F. A.	Hagen
Danielson, Lennox	Litchfield	Engstrom, G. F.	Hagen
Darling, J. B.	St. Paul	Eppard, R. M.	Hagen
Darnall, C. M.	Rochester	Erb, F. A.	Hagen
Dart, L. O.	Minneapolis	Erdman, C. A.	Hagen
Daugherty, E. B.	St. Paul	Erickson, Eskil	Hagan
Daugherty, L. E.	St. Paul	Erickson, R. F.	Hanson
Davis, A. C.	Rochester	Erickson, J. G.	Hanson
Davis, B. F.	Duluth	Erickson, R. M.	Hanson
Davis, F. U.	Faribault	Erickson, Swan	Hanson
Davis, Herbert	St. Paul	Ernest, G. C.	Hanson
Davis, I. G.	Rushford	Eshelby, E. C.	Hanson
Davis, William	St. Paul		Hanson
De Boer, Hermanus	Edgerton		Hanson

\*Deceased

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Ghormley, R. K.	Rochester	Harrington, C. D.	Minneapolis	Holl, P. M.	Minneapolis
Ghostley, Mary C.	Pupsoky	Harrington, F. E.	Minneapolis	Hollands, W. H.	Fisher
Gibbs, E. C.	St. Paul	Harrington, S. W.	Rochester	Holm, H. H.	Glencoe
Gibson, G.	Rochester	Harris, C. N.	Hibbing	Holm, P. F.	Wells
Giere, E. O.	Minneapolis	Hart, W. E.	Monticello	Holmberg, L. J.	Canby
Giere, J. C.	Minneapolis	Hartiel, W. F.	St. Paul	Holmes, A. E.	Rush City
Giere, R. W.	Minneapolis	Hartley, E. C., Jr.	St. Paul	Holmstrom, C. H.	Warren
Giere, S. W.	Benson	Hartman, H. R.	Rochester	Holst, C. F.	Little Falls
Giesen, A. F.	Starbuck	Hartzell, T. B.	Minneapolis	Holst, J. B.	Little Falls
Gessler, P. W.	Minneapolis	Haskell, A. D.	Alexandria	Holt, W. B.	Minneapolis
Giffin, H. Z.	Rochester	Hassett, R. G.	Mankato	Holt, J. E.	St. Paul
Gilliland, J. S.	St. Paul	Hastings, D. R.	Minneapolis	Holtan, Theodore	Waterville
Gilles, F. L.	Minneapolis	Hatch, W. E.	Duluth	Holte, Halvor	Crookston
Gillespie, J. B.	Rochester	Hathaway, S. J.	Proctor	Horton, B. T.	Rochester
Gillespie, M. G.	Duluth	Hauge, M. I.	Clarkfield	Hospodarsky, L. J.	New Prague
Gillespie, N. H.	Duluth	Hause, V. P.	St. Paul	Hottinger, R. C.	Janesville
Gilmore, Rowland	Bemidji	Havens, F. Z.	Rochester	House, Z. E.	Cass Lake
Gilpin, S. F., Jr.	Rochester	Havens, J. G. W.	Austin	Howard, Laura K.	Fergus Falls
Ginsberg, Harry	Minneapolis	Haeverfield, A. R.	Minneapolis	Howard, M. I.	Mankato
Ginsberg, William	St. Paul	Hawkins, V. J.	St. Paul	Howard, W. H.	Minneapolis
Gobirsch, A. P.	Sleepy Eye	Hawkinson, L. F.	Brainerd	Howard, L. P.	St. Paul
Goehrs, H. W.	St. Cloud	Hawkinson, R. P.	Robbinsdale	Howell, W. L.	Rochester
Golden, C. M.	Tyler	Hayes, J. M.	Minneapolis	Hubbard, O. E.	Brainerd
Goldish, D. R.	Duluth	Hayes, M. F.	Nashwauk	Hubin, E. G.	Deerwood
Goltz, E. V.	St. Paul	Haynes, A. L.	Faribault	Huenekens, E. J.	Minneapolis
Goodwin, T. W.	Rochester	Head, D. P.	Minneapolis	Huffington, H. L.	Mankato
Gowan, L. R.	Duluth	Head, G. D.	Minneapolis	Hughes, L. D.	Minneapolis
Graham, David	Duluth	Healy, R. T.	Pierz	Hulkkrans, J. C.	St. Paul
Graham, R. D.	Duluth	Hearn, W. O.	Minneapolis	Hultkrans, R. E.	Minneapolis
Graham, Robert	Duluth	Heath, A. C.	St. Paul	Humphrey, E. W.	Moorhead
Graham, W. D.	Hanska	Hebeisen, M. B.	Chaska	Humphrey, W. R.	Stillwater
Grant, H. W.	St. Paul	Hebert, W. H. J.	Rochester	Hunt, F. N.	Fairmont
Gratzek, F. R.	Minneapolis	Heck, F. J.	Rochester	Hunt, R. C.	Fairmont
Gratzek, Thomas	St. Paul	Heck, W. W.	St. Paul	Hurd, Anna	Minneapolis
Grave, Floyd	Minneapolis	Hedback, A. E.	Minneapolis	Husband, M. W.	Minneapolis
Graves, W. N.	Duluth	Hedberg, G. A.	Popeming	Huseby, H. W.	Floodwood
Grawn, F. A.	Northome	Hedenstrom, F. G.	St. Paul	Hutchinson, Henry	New London
Gray, F. D.	Marshall	Hedenstrom, L. H.	Cambridge	Huxley, F. R.	Faribault
Gray, H. K.	Rochester	Hedin, R. F.	Red Wing	Hyde, T. L.	Rochester
Greene, E. K.	Minneapolis	Hegge, O. H.	Austin	Hynes, J. E.	Minneapolis
Greene, H. H.	Austin	Hegge, R. S.	Austin	Iber, F. C.	Rochester
Greene, W. P.	Minneapolis	Heiberg, E. A.	Fergus Falls	Ide, A. W.	St. Paul
Grimes, A. V., Jr.	Rochester	Heim, R. R.	Minneapolis	Ikeda, Kano.	St. Paul
Grimes, H. B.	Madelia	Heimark, J. H.	Moorhead	Imes, P. R.	Rochester
Grimes, Marian	Minneapolis	Heimark, J. J.	Fairmont	Irwin, A. F.	Minneapolis
Grinnell, W. B.	Preston	Heimark, O. E.	Duluth	Jackson, C. M.	Minneapolis
Grise, W. B.	Austin	Heise, W. F. C.	Winona	Jacobs, A. C.	Elmore
Groschupf, T. P.	Bemidji	Heik, H. H.	Minneapolis	Jacobs, G. C.	Fergus Falls
Grose, F. N.	Clarissa	Helland, G. M.	Spring Grove	Jacobs, J. C.	Willmar
Gruenhagen, A. P.	St. Paul	Helland, J. W.	Spring Grove	Jacobson, Clarence.	Chisholm
Gullixson, A.	Albert Lea	Helmholtz, H. F.	Rochester	Jacobson, D. J.	Blackduck
Gully, R. J.	St. Peter	Hempstead, B. E.	Rochester	Jacquot, G. L.	Marshall
Gundersen, N. A.	Minneapolis	Hemstead, Werner	St. Cloud	Jamieson, Earl.	Walnut Grove
Gustafson, H. T.	Minneapolis	Hench, P. S.	Rochester	Jennings, F. L.	Oak Terrace
Habein, H. C.	Rochester	Henderson, A. J.	Kiester	Jennings, Mary H.	Minneapolis
Haberman, E.	Oskakis	Henderson, M. S.	Rochester	Jensen, A. H.	Hutchinson
Hacking, F. H.	Minneapolis	Hendrickson, J. F.	Minneapolis	Jensen, H. H.	Atwater
Haddow, N. W.	Minneapolis	Hendrickson, R. R.	Wadena	Jensen, Harry.	Minneapolis
Hagaman, G. K.	St. Paul	Hengstler, W. H.	St. Paul	Jensen, M. J.	Minneapolis
Hagen, G. L.	Minneapolis	Henney, W. H.	McIntosh	Jensen, T. J.	Duluth
Hagen, H. O.	New Richland	Henry, C. E.	Minneapolis	Johnson, A. B.	Minneapolis
Hagen, O. E.	Butterfield	Henry, M. O.	Minneapolis	Johnson, A. E.	Minneapolis
Hagen, O. I.	Moorhead	Hengstler, W. H.	St. Paul	Johnson, A. M.	Red Wing
Haessly, S. B.	Faribault	Henney, W. H.	McIntosh	Johnson, C. E.	Cambridge
Haggard, G. D.	Minneapolis	Hensel, C. N.	St. Paul	Johnson, C. M.	Dawson
Haight, G. D.	Minneapolis	Henslin, A. E.	Le Roy	Johnson, E. W.	Bemidji
Haines, J. H.	Audubon	Herbert, W. L.	Maynard	Johnson, Ellsworth	Rochester
Haines, S. F.	Stillwater	Herman, A. L.	Minneapolis	Johnson, H. C.	St. Paul
Hale, D. E.	Rochester	Hermann, P. E.	Hendricks	Johnson, H. M.	Dawson
Halgren, H. A.	Rochester	Hermann, E. T.	St. Paul	Johnson, H. P.	Fairmont
Hall, A. R.	Watertown	Hertel, G. E.	Austin	Johnson, Hans.	Kerkhoven
Hall, H. H.	St. Paul	Hesdorfer, M. B.	Minneapolis	Johnson, J. A.	Minneapolis
Hall, J. M.	Minneapolis	Hesselgrave, V. G.	Taylors Falls	Johnson, J. A.	St. Paul
Hallenbeck, C. A.	Minneapolis	Hesseltine, S. S.	St. Paul	Johnson, Julius.	Minneapolis
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Hammes, E. M.	St. Paul	Hilger, D. D.	St. Paul	Johnson, R. G.	St. Paul
Hammond, A. J.	Minneapolis	Hilger, L. A.	St. Paul	Johnson, S. M.	Minneapolis
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Hanover, R. D.	Littlefork	Hirschfelder, F. R.	Minneapolis	Johnson, W. R.	Rochester
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Hansen, E. W.	Minneapolis	Hoaglund, A. W.	Minneapolis	Johnson, Y. T.	Minneapolis
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Harper, F. R.	Rochester	Holcomb, J. T.	St. Paul		

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Klein, Harry	Duluth	Lippmann, E. W.	Hutchinson	McGrawan, H. T.	Red Wing
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Kvitrud, G.	St. Paul	Lymburner, R. M.	Rochester	Meierding, W. A.	New Ulm
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Lamb, H. L.	Little Falls	Lynes, Henry	Minneapolis	Mellby, O. F.	Thief River Falls

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Newell, C. E.	Rochester	Nordin, G. T.	Rochester	Rothschild, H. J.	St. Paul
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